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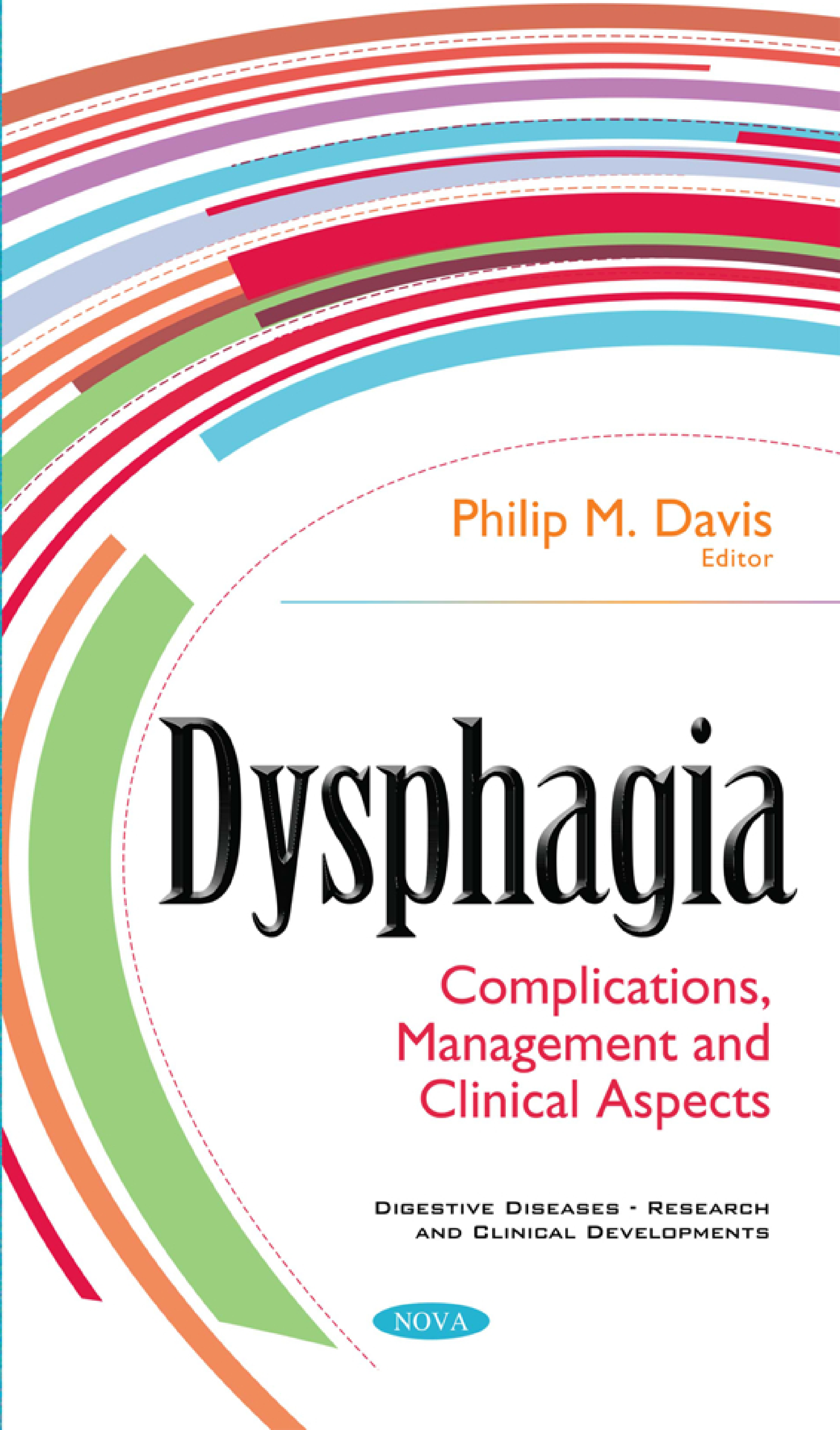

Philip M. Davis
Editor

Dysphagia

Complications, Management and Clinical Aspects

DIGESTIVE DISEASES - RESEARCH
AND CLINICAL DEVELOPMENTS

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DYSPHAGIA

COMPLICATIONS, MANAGEMENT AND CLINICAL ASPECTS

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COMPLICATIONS, MANAGEMENT AND CLINICAL ASPECTS

PHILIP M. DAVIS
EDITOR

New York

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PREFACE

Dysphagia is the difficulty or improper swallowing of liquids, solids, or even saliva. This book provides new research on the complications, management and clinical aspects of dysphagia. Chapter One reviews the development of normal swallowing in infants and children and protective reflexes to prevent aspiration; the pathophysiologic events that occur due to aspiration and some of the disease processes in children associated with dysphagia; and current methods of diagnosis of dysphagia and its therapy in children. Chapter Two focuses on dysphagia after suffering a stroke. Chapter Three discusses the incidence, mechanism, and treatment of dysphagia and pneumonia in acute intracerebral hemorrhage. Chapter Four reviews the epidemiology of dysphagia in patients with Duchenne muscular dystrophy (DMD) and the method of evaluating their swallowing problems.

Chapter 1 – Dysphagia in children, particularly if it leads to aspiration, is a significant cause of respiratory morbidity and sometimes mortality. Aspiration occurs in the setting of dysphagia when airway protective reflexes fail. Clinical symptoms and physical findings of dysphagia and aspiration in children can be nonspecific. However, advances in technology can lead to early diagnosis, and new therapeutic modalities can improve outcome and prognosis. In this chapter, the development of normal swallowing in infants and children and airway protective reflexes to prevent aspiration will first be reviewed. Next, the pathophysiologic events that occur due to aspiration and some of the disease processes in children associated with dysphagia will be reviewed. Finally, the current methods of diagnosis of dysphagia and its therapy in children will be reviewed.

Chapter 2 – Dysphagia is a common consequence after stroke, presenting in up to 50% of all acute stroke patients, with increased morbidity and mortality mainly because it dramatically increases the risk of aspiration pneumonia, and because it leads to malnutrition and dehydration as well. It has been reported that the incidence of dysphagia ranges from 25% to 81% depending on the timing of the assessment, diagnostic methods and criteria. The nervous system affected by a stroke damage is thought to reserve a variable ability to somehow reorganize damaged pathways and structures, allowing an effective recovery from swallowing impairment. Notwithstanding, about half of stroke patients remain dysphagic after seven days from symptoms onset, and approximately 13-15% of patients has persistent swallowing dysfunction after six months, with a dramatic impact on patients' outcome in terms of increased likelihood of residential placement and estimated lifetime costs in stroke survivor. To date, it is unclear why some stroke patients develop a persistent swallowing impairment after stroke or, in other words, if the failure in recovery from dysphagia is due to a stroke involving the dominant swallowing hemisphere, rather than a swallowing key region, or even to any specific clinical or neuroradiological stroke factor. In this view, stroke severity upon admission, assessed using NIHSS, is an important clinical predictor of post-stroke dysphagia and of a persistent pattern of dysphagia as well. Regarding the site of the stroke lesion, it is widely accepted that swallowing function is provided by a complex neural network bilaterally involving the perirolandic sensorimotor cortex which, together with other cortical structures, projects and receives afferences to the brainstem' Central Pattern Generator, through the basal ganglia, the thalamus and the periventricular white matter as well. Despite several cortical areas have been described as related with swallowing impairment, a definite role in causing dysphagia has been not completely understood for all of them so far. Interestingly, recent studies reported that the disruption of cortical-cortical and cortical-subcortical white matter connections seems to increase the risk of dysphagia and aspiration pneumonia by lowering the threshold of input to the medullary swallowing center. Thus, dysphagia may result from the combined effects of an acute focal damage and a preexisting impairment of the subcortical white matter connecting pathways. In other words, the authors might think of dysphagia as a kind of deficit in executive functions, which they know to be particularly associated with leukoaraiosis and cognitive disorders, and of course age.

Chapter 3 – Dysphagia, defined as difficulty in the passage of solids or liquids from the mouth to the stomach, is common after stroke. Aspiration is a major risk factor for the development of pneumonia. Dysphagia and pneumonia in intracerebral hemorrhage (ICH), however, have not been well investigated. Incidence, mechanism, and treatment of dysphagia and pneumonia in acute ICH are discussed.

Chapter 4 – Duchenne muscular dystrophy (DMD) is the most common childhood onset muscular dystrophy, with an incidence estimated as 1:3000 live male births. It is an X-linked inherited condition, caused by mutation of the dystrophin gene. Although much of the literature on DMD states that the disease impacts on swallowing function and recommends swallowing assessment as part of their routine examination, the appropriate method of evaluating their swallowing problems had not yet been determined. Recently, the authors developed an 8-stage Neuromuscular Disease Swallowing Status Scale (NDSSS) and demonstrated its sufficient reliability, validity, and responsiveness in 134 patients with DMD. NDSSS is considered as a useful tool to evaluate swallowing disorders in clinical area, because it is easy to understand and administer by either medical or nonmedical staff. The authors recommend that their swallowing function should be evaluated periodically and followed up using the NDSSS, and timely consider more detailed assessment with videofluorography. In this chapter, they reviewed the epidemiology of dysphagia in patients with DMD and the method of evaluating their swallowing problems.

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Chapter 1

PEDIATRIC DYSPHAGIA AND ASPIRATION: EVALUATION AND MANAGEMENT

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ABSTRACT

Dysphagia in children, particularly if it leads to aspiration, is a significant cause of respiratory morbidity and sometimes mortality. Aspiration occurs in the setting of dysphagia when airway protective reflexes fail. Clinical symptoms and physical findings of dysphagia and aspiration in children can be nonspecific. However, advances in technology can lead to early diagnosis, and new therapeutic modalities can improve outcome and prognosis. In this chapter, the development of normal swallowing in infants and children and airway protective reflexes to prevent aspiration will first be reviewed. Next, the pathophysiologic events that occur due to aspiration and some of the disease processes in children associated with dysphagia will be reviewed. Finally, the current

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methods of diagnosis of dysphagia and its therapy in children will be reviewed.

INTRODUCTION

Dysphagia, difficult or improper swallowing of liquids, solids, or even saliva, is felt to affect approximately 1% of children in the United States annually [1]. Dysphagia can lead to aspiration, the inhalation of foreign material into the lower airways. Aspiration can be an acute event or a chronic syndrome. When it is chronic and recurrent in children, the effects on lung development can be devastating and lead to significant respiratory problems that can severely impair pulmonary function and can lead to pulmonary scarring. Thus, aspiration can lead to significant morbidity and it can also occasionally lead to death. To prevent or minimize this, the diagnosis of dysphagia and aspiration needs to be made as early as possible during life. However, dysphagia and aspiration frequently go unrecognized by primary care physicians or caregivers as a cause of chronic respiratory symptoms in children [2].

In this chapter, the development of normal swallowing in infants and children and protective reflexes to prevent aspiration will first be reviewed. Next, the pathophysiologic events that occur due to aspiration and some of the disease processes in children associated with dysphagia will be reviewed. Finally, the current methods of diagnosis of dysphagia and its therapy in children will be reviewed.

THE DEVELOPMENT OF SWALLOWING

Development of swallowing begins early during fetal life and this development is integrated with development of the respiratory centers of the brainstem. Pharyngeal swallowing can be detected by ultrasound between 10 and 14 weeks of gestation. Nonnutritive sucking and swallowing occurs at 15 weeks of gestation and sucking with anterior to posterior tongue movements between 18 and 24 weeks of gestation. Nonnutritive sucking on a pacifier may be observed in the 26 to 29 weeks of gestation infant. Some infants can feed orally at 32 to 33 weeks of gestation but, the earliest that an infant can sustain full oral nutrition and hydration is 34 weeks of gestation [3]. Postnatally, at approximately 4 to 6 months of age, an infant can consume strained or pureed foods orally. Increased food textures can be given to infants at age 7 to 9 months as head control and stability and differentiation of tongue movements improve. With further feeding development, the infant can be transitioned to table foods at about 1 year of age. By the age of 2 years, oral motor, feeding, and swallowing skills should be in place [4].

The swallowing mechanism is part of the upper aerodigestive tract which includes the oral, pharyngeal, and nasal cavities and the larynx. The position, size, and shape of these structures in infants provide the optimal arrangement functionally for safe, effective nipple feeding. Between the ages of approximately 5 months and 3 years, the larynx, which is located at the level of the first to third cervical vertebrae in the newborn, descends in the pharynx to the level of the sixth to seventh cervical vertebrae, resembling the aerodigestive anatomy of the adult. As the larynx descends, it allows for greater variety of vocalizations. Also, as the larynx descends, increased neuromuscular control of the structural elements of the pharynx must occur to maintain airway protection during swallowing [2, 4].

The oral, nasal, pharyngeal, and laryngeal structures are primarily innervated by branches of cranial nerves I, V, VII, IX, X, XI (vagal branch), XII, and branches of the upper cervical nerves. Afferent input for swallowing is primarily located in the nucleus tractus solitarius; efferent control is localized in the nucleus ambiguous and the dorsal nucleus of cranial nerve X [4]. Swallowing can be divided into three distinct phases: oral (to include both oral preparatory and oral transit phases), pharyngeal, and esophageal. Each of the three phases plays a part in directing a bolus of salivary secretions or ingested food into the esophagus, and not into the air passages [5].

AIRWAY PROTECTIVE REFLEXES

The airway is protected from aspiration by a series of reflexes with sensors in the pharynx, larynx, and esophagus. These mechanoreceptors, thermal receptors, baroreceptors and chemoreceptors are concentrated over the surface of the pharynx, epiglottis, arytenoids cartilages, and vocal cords [6, 7]. The chemoreceptors are activated by water, as well as a variety of salts, sugars, and acids [8, 9]. Activation of this reflex results in glottis closure and inhibition of respiration with or without swallowing [4]. Failure of these protective reflexes allows aspiration to occur from swallowed boluses or material refluxed up the esophagus from the stomach [2].

The nature of the protective reflex invoked varies as a function of the age of the individual and the region of the pharynx or larynx that is stimulated. Mechanical stimulation of pharyngeal afferent receptors stimulates swallowing at any age. In susceptible human infants, stimulation of the laryngeal chemoreflex by acid, water, or milk can lead to prolonged apnea rather than coughing and swallowing [2]. This developmental influence is most likely due to central respiratory inhibition [10]. Viral respiratory infections, such as respiratory syncytial virus, which causes increased upper airway secretions, can cause the adult response pattern, that is, coughing and swallowing, to revert to the immature pattern in which apnea predominates [11]. The apnea, which can last longer than 30 seconds, is first central and then obstructed (i.e., mixed) and usually terminates after a series of swallows, or occasionally a cough [12, 13].

Maturation of the laryngeal cough reflex leads to an increase in coughing and a decrease in swallowing and apnea. These changes have been attributed to central processing of afferent (sensory) stimuli as opposed to a reduction in the sensitivity of the larynx or any major change in the distribution or quantity of receptor sites within the larynx. Laryngeal chemoreceptors in the newborn are the primary defense against aspiration of liquids. As the infant matures, the laryngeal cough reflex becomes more prominent. In adults, the chemoreflexive responses of the larynx continue to provide the primary source of airway protection [14].

PATHOPHYSIOLOGIC EVENTS ASSOCIATED WITH ASPIRATION

Aspiration can cause permanent damage to the developing lungs of infants and children. Aspiration of large particles can cause acute airway obstruction and severe hypoventilation. Smaller particles or liquid aspirates may induce hypoxemia by a variety of mechanisms, including reflex airway closure, hemorrhagic pneumonitis, destruction and dilution of surfactant with secondary atelectasis, and pulmonary edema from extravasation of intravascular fluids and protein [15]. As the pH of aspirated material drops below 2.5, lung injury increases, with maximal lung injury occurring at a pH of 1.5 [16]. The volume of aspirated material plays a major role in lung injury. In dogs, 1 ml/kg of acid aspiration produced only mild effects in the lungs, whereas 2ml/kg or more of acid aspiration caused serious effects in the lungs, usually death [17]. Histologic findings of aspiration include degeneration of bronchiolar epithelium, pulmonary edema and hemorrhage, focal atelectasis, exudation of fibrin, and acute inflammatory cell infiltrate. Gastric contents instilled into the trachea of dogs appeared on the lung surface within 12-18 seconds. Extensive atelectasis developed within 3 minutes. Changes of acute pneumonia occurred within hours, and granulomatous changes developed within 48 hours [18, 19]. Chronic pulmonary aspiration can ultimately lead to the development of bronchiectasis in children [20].

CAUSES OF DYSPHAGIA IN CHILDREN

Aspiration is most commonly the result of dysphagia, gastroesophageal reflux disease, or insufficient management of nasal/oral secretions. There are numerous conditions that predispose children to aspiration lung injury. As shown in the Table, these conditions can be subdivided into anatomic, neurologic, gastrointestinal, and other causes. Several syndromes also predispose children to aspiration. Chronic aspiration secondary to dysphagia most often occurs secondary to neurologic disease, functional immaturity, or anatomic causes [3].

Table. Conditions Predisposing to Aspiration in Children

Anatomic
Choanal stenosis
Cleft lip/palate
Laryngotracheal cleft
Esophageal atresia
Tracheoesophageal fistula
Craniofacial abnormalities
Vascular ring
Tumors
Cystic hygroma
Laryngomalacia
Tracheal stenosis
Laryngeal/pharyngeal trauma
Laryngeal/pharyngeal vascular malformations
Lingual/palatine tonsillar hypertrophy

Table. (Continued)

Syndromes
Pierre-Robin
Beckwith-Wiedemann
Down (sometimes)
CHARGE
Pfeiffer
Cri du chat
Neuromuscular
Perinatal asphyxia
Cranial nerve or recurrent laryngeal nerve injury
Congenital hydrocephalus
Neonatal intraventricular hemorrhage
Dysautonomia
Moebius syndrome
Myotonic dystrophy
Wernig-Hoffman disease
Cornelia de Lange syndrome
Muscular dystrophy
Myasthenia gravis
Guillian-Barre syndrome
Cerebral palsy
Vocal cord paralysis/dysfunction
Arnold-Chiari malformation (sometimes)
Depressed consciousness
Static/progressive encephalopathy
Traumatic brain injury
Cerebrovascular accident
Hydrocephalus
Ataxia telangectasia
Gastrointestinal
Gastroesophageal reflux
Esophageal motility dysfunction
Malrotation
GI obstruction/stricture
Achalasia

Other
Developmental/immaturity of swallowing
Respiratory syncytial virus bronchiolitis
Endotracheal tubes/tracheostomy tubes/postextubation
Foreign body aspiration
Collagen vascular disease
Obstructive sleep apnea
Bottle-propping

Premature infants are at significant risk for dysphagia. If there is incomplete development or coordination of the suck-swallow-breathe mechanism, aspiration due to dysphagia can occur [21, 22]. Often this problem occurs in association with a lingering neonatal respiratory illness with or without the presence of tachypnea [23-25]. Occasionally, infants born at term may have dysphagia, but this seems to improve over time with growth and further neurological development [26].

Dysphagia with aspiration can occur in young infants due to fatigue of the swallowing mechanism if they are pushed to take prolonged or large feedings, particularly toward the end of the feedings [3]. Infants placed in their beds with bottles propped in their mouths are at risk for dysphagia and aspiration, especially when they try to suck and swallow when only partially awake [27].

Anatomic problems involving the nasopharynx, oropharynx, and trachea can result in dysfunctional swallowing with resultant aspiration [2]. In infants with choanal stenosis who develop respiratory distress during oral feedings, increased nasal resistance to airflow and the infant's necessity for nasal breathing cause the infant to be unable to adequately coordinate sucking and swallowing with breathing [28]. Infants with cleft lips and palates are at risk for aspiration due to nasal reflux of feeding causing them to gasp and have aspiration [28]. Also, aspiration may occur if food in the nasal cavity falls into a still open airway after the infant has swallowed [2]. Infants with laryngotracheal clefts, esophageal atresia, and tracheoseophageal fistulas are at risk for respiratory difficulties, such as coughing, choking, cyanosis, or respiratory distress in association with feeding, and feeding may precipitate aspiration with the subsequent risk of recurrent pneumonia [29]. In infants and children with certain craniofacial abnormalities, the presence of micrognathia results in relative macroglossia that compromises the oral airway and often interrupts, impairs, or prevents successful oral feeding [3]. Laryngomalacia, resulting in persistent or intermittent upper airway obstruction can disrupt the timing of the swallow [3]. Vascular rings and pulmonary slings are well-defined congenital anomalies of the aortic arch, brachiocephalic arteries, or pulmonary arteries that cause symptoms due to compression of the airway, esophagus, or both. Respiratory symptoms, such as stridor, barking cough, and wheezing predominate, but dysphagia may be present as well [30]. Neck cysts and tumors, such as cystic hygromas, lymphatic malformations, neuroblastomas, and hemangiomata, may narrow the upper aerodigestive tract as well as impair the movements of laryngeal and pharyngeal structures of deglutition [3].

Infants with Down syndrome have protruding tongues and occasionally cleft palates or tracheoesophageal fistulas that can put them at risk for feeding difficulties and aspiration [2]. Some patients with Down syndrome who have respiratory difficulty and stridor have been reported to have midtracheal stenosis [31]. This would seem to put them at high risk for aspiration [2]. Congenital syndromes that result in lower cranial nerve dysfunction such as Moebius syndrome and CHARGE association often have dysphagia complicated by chronic aspiration [3].

Infants and children with neurologic or neuromuscular dysfunction may develop aspiration. They may be unable to feed by nipple or at the breast due to an inability to coordinate breathing and swallowing, or because they lack adequate bulbar muscle function to successfully feed [28]. They may have prolonged feeding time due to poor oral preparation, neck extension impairing laryngeal elevation, premature spillage of liquids, poor pharyngeal clearance with excessive residual, and poor esophageal opening [3]. They also may have a weak gag mechanism or ineffective cough that predisposes them to acute and chronic aspiration [32]. Aspiration is the most common cause of death in these patients. Choking with feeding, chest infections, and dysphagia may be present in nearly all children with severe cerebral palsy. Children with Arnold-Chiari malformation, due to compression of the brainstem and subsequent effect on lower cranial nerve function, may have pharyngoesophageal dysfunction and cricopharyngeal achalasia due to delayed passage of a food bolus into the esophagus and increased pooling of food and oral secretions in the hypopharynx. They may have unilateral or bilateral vocal cord dysfunction or paralysis that interferes with coordination of respiration and swallowing and impairs protective posterior glottis closure [3]. Infants who have a difficult vaginal delivery of their heads with the possibility of a cranial nerve or laryngeal recurrent nerve injury, or infants who have perinatal asphyxia, congenital hydrocephalus, or neonatal intraventricular hemorrhage need to be screened for the presence of feeding difficulties by a feeding team with subsequent testing for dysphagia prior to their initial attempt at oral feeding [28].

Aspiration can occur in infants and children with endotracheal tube placement or with tracheostomies. Endotracheal tubes can cause remodeling of the palate due to pressure of the tube on the hard and soft palate. After extubation, the infant may be unable to establish a seal around the nipple during feeding due to plalato-pharyngeal incompetence, leading to aspiration secondary to nasal reflux of feedings or defective integration of the sucking and swallowing mechanism [33]. The presence of a tracheostomy tube interferes with the swallowing mechanism by preventing the subglottic rise in tracheal pressure, altering the timing of the swallow, and limiting elevation of the larynx during swallowing [3, 34-36]. The use of a one-way speaking valve has been shown to improve swallowing function in adults with a tracheostomy by improving oral and pharyngeal sensation, restoring physiologic positive end-expiratory pressure (PEEP), and increasing subglottic pressure during swallowing [2, 3]. The use of these speaking valves have been shown to decrease the occurrence of aspiration in adults [37].

SYMPTOMS OF ASPIRATION

Children with chronic aspiration can present to their physicians with complaints such as wheezing that is poorly responsive to appropriate therapies, chronic cough, recurrent pneumonia, atelectasis, bronchiectasis, pulmonary abscess, pulmonary fibrosis, bronchiolitis obliterans, apnea/bradycardia/acute life-threatening events, failure to thrive, stridor, or laryngitis/hoarseness [2]. Infants and children with an absent or ineffective cough reflex may have silent aspiration and have findings of only increased respiratory mucus, congestion and chronic wheeze or rhonchi, recurrent bronchitis, or recurrent pneumonia [28, 38].

PHYSICAL EVALUATION OF ASPIRATION

The clinician should observe the infant or child eating and auscultate the chest and back both before and after feeding for crackles, wheezes, “wet” upper airway noises, and “wet” voice quality. During feeding, attention should be given to nasopharyngeal reflux, difficulty when sucking or swallowing, and associated coughing and choking. Drooling or excessive accumulation of secretions in the mouth suggests salivary dysphagia [39].

RADIOLOGIC EVALUATION OF ASPIRATION

The chest radiograph is often the first test performed by the clinician for an infant or child with chronic or recurrent respiratory symptoms. In a study of 22 children with recurrent aspiration, the chest radiograph was normal in 14% and revealed only bronchial wall thickening or hyperinflation in 18%. The radiographs in the rest of the children revealed diffuse (27%) or localized (41%) infiltrates. Infiltrates in infants with recurrent aspiration are often in dependent areas such as the upper lobes and the posterior areas of the lower lobes [32]. Chest radiographs are generally insensitive to early changes of lung injury [40]. Computed tomography scans, particularly high-resolution images, are more sensitive in the detection of early airway and parenchymal disease in children who aspirate [41]. Findings of bronchial wall thickening, air-trapping, bronchiectasis, ground-glass opacities, and centrilobular opacities (“tree in bud”) are common in children who aspirate chronically [42].

DIAGNOSIS OF DYSPHAGIA IN INFANTS AND CHILDREN

The pediatric assessment for dysphagia begins with a thorough review of the patient’s history, including medical, developmental, and feeding history [41, 43-45]. Clinical feeding assessment, usually by a trained speech therapist, includes observation of the parent and child during feeding, oral peripheral examination, communicative behavior, and observations of the child before and after feeding [43-45]. This assessment may also include a screening test for aspiration such as the 3-ounce water swallow challenge [46]. In one study, the sensitivity of this test for predicting aspiration in children from 2 to 18 years of age was 100% with a specificity of 51%. The sensitivity and specificity of this test for predicting which children could safely intake thin oral liquids was 100% and 44%, respectively. However, the test, due to a high false positive rate and low specificity may not be the best screening tool for identifying children who are at risk for aspirating thin oral liquids [47].

Instrumental assessment is necessary for definitive diagnosis of swallowing dysfunction and aspiration [48]. This assessment may include use of the videofluoroscopic swallow study (VFSS), also commonly known as the modified barium swallow study (MBS) or fiberoptic endoscopic evaluation of swallowing with or without sensory testing (FEES or FEESST) [43-45, 49]. Each procedure has strengths and weaknesses [2]. Neither test has been established as a "gold standard" for the detection of aspiration [2].

The VFSS provides visualization of the anatomy of the oral cavity, pharynx, larynx, and upper esophagus as well as the function and integration of all four areas during the dynamic process of swallowing. It provides the most thorough assessment of swallowing function and of compensatory measures to improve swallowing function in the pediatric population. It is generally considered to be a reliable and safe method of diagnosing dysphagia in the pediatric population. The ultimate goal of the VFSS is to determine the physiologic cause of the dysphagia symptom (aspiration or laryngeal penetration, nasopharyngeal backflow, swallow trigger, and pharyngeal residual) and then identify the most appropriate treatment strategies to allow the safe and most appropriate intake of calories for infants and children [2]. The test does have several disadvantages. Its accuracy can be affected by patient cooperation due to the necessary addition of barium to food and liquids. Infants and children who only have negligible amounts of oral intake are not appropriate candidates for VFSS as a sufficient amount of contrast must be ingested to produce adequate imaging [4]. The test employs a significant amount of radiation exposure to the brain that could pose risk to small infants or in children with significant brain injuries [2]. In adults, as compared to FEES, the sensitivity of detecting aspiration using VFSS is 100% with a specificity of 63% [2, 50-53].

Swallowing function can also be evaluated by FEES. This evaluation is often performed by a trained otolaryngologist and a speech therapist working together. FEES requires no radiation exposure or sedation of the patient [3]. The food or liquids that the child consumes during the test are colored with food dye but are not altered by the addition of barium [2, 4]. The specific focus of FEES is evaluation of airway protection during swallowing [4]. FEES enables direct visualization of soft tissue structures before and after the swallow [42, 54]. During feeding, FEES can identify oropharyngeal dysfunction that occurs before the swallow such as spillover and laryngeal penetration or aspiration. After the swallow, residual material can be seen, and the presence of laryngeal penetration or aspiration from the residue can be noted [2]. Additionally, aspiration during the swallow may be identified with visualization of food or liquid in the trachea after the swallow [55]. There are some disadvantages to the pediatric FEES examination. There is possible discomfort with endoscope passage into the airway. Patients with oral hypersensitivity or oral aversion may have a gagging reaction in response to the tactile stimulation of the instrument. The FEES exam is limited to the pharyngeal phase of swallowing. There is a loss of view or “white out” during the swallow caused by pharyngeal contraction and subsequent light deflection. FEES is contraindicated in patients with choanal atresia, nasal stenosis, nasal obstruction, or pharyngeal stenosis [4]. In adults, as compared to VFSS, the sensitivity for detecting aspiration is 70%-96% with a specificity of 87.5%-100% [50, 56-60].

FEESST gives the examiner information about swallowing dysfunction and about the laryngeal adductor reflex (LAR). The LAR, which is vital for airway protection, is stimulated by providing controlled air pulses to the aryepiglottic folds through the port of a flexible laryngoscope. By stimulating them with increasing pressure in the form of the air pulse, the examiner gains information about laryngopharyngeal mechanosensitivity. Normal LAR is elicited with <4.0 mmHg of air pulse pressure; anything greater than that is considered abnormal [61-65]. This allows FEES to evaluate children who may aspirate oral secretions or to assess a non-orally feeding child’s safety to begin eating by mouth [3].

OTHER DIAGNOSTIC TESTS OF ASPIRATION

Some children may have aspiration of saliva with continued respiratory symptoms even if other causes of aspiration from oral feeding and gastroesophageal reflux have been effectively treated. The radionuclide salivagram is used to try to detect salivary aspiration. A small quantity of radiotracer is placed in the buccal pouch, and serial images are taken until the tracer clears the mouth. Activity in the trachea or bronchi indicates aspiration. Unfortunately, the ability of the salivagram to detect salivary aspiration in children is only in the range of 16% to 39% [66-69]. Salivagrams also have poor correlation with VFSS for detecting aspiration [70]. Thus, while the salivagram is useful for the detection of aspiration in some patients, further studies need to be conducted to evaluate its sensitivity and specificity. However, there is no “gold standard” exam for detection of salivary aspiration to which the salivagram can be compared [2]. In some centers, if a child suspected of salivary aspiration has a negative salivagram, FEES or FEESST is often then performed [3].

Several biomarkers have been suggested as indicators of aspiration. These include pepsin, bile acids, and milk protein. Of these, pepsin has been studied the most. Pepsin, an acidic protease, is the primary proteolytic enzyme of the digestive system. It is not known to be produced by extraesophageal tissues and it is a measure of gastroesophageal reflux if found in the upper airway and reflux aspiration if found in the lungs [71]. Pepsin is produced when pepsinogen is cleaved to pepsin in an acidic environment. Pepsinogen is produced by the chief cells of the stomach lining [71]. However, pepsinogen is also produced in the lung and is found in serum. Thus, activation of pepsin by acidification can also potentially cleave pepsinogen to pepsin leading to false positive results when measured by enzymatic assays such as ELISA and Western blotting techniques [71].

To attempt to detect reflux aspiration in adults and children, pepsin levels are measured in airway fluid obtained by bronchoalveolar lavage, saliva, or exhaled breath condensate [71]. In one study, pepsin enzymatic activity was detected in 31 of 37 tracheal aspirates from children with gastroesophageal reflux compared with 0 of 26 healthy controls [73]. Significantly raised bronchoalveolar lavage pepsin levels have also been found in children with chronic cough and proximal reflux [74]. In a study of 96 children with chronic lung disease, with, and without gastroesophageal reflux, the bronchoalveolar lavage pepsin level was raised. However, specificity was low, and there was extensive group overlap [75]. Pepsin has been used as a marker of tracheal aspiration in children ventilated in intensive care units. However, results vary widely with reported pepsin positivity in tracheal aspirate ranging from 13.5% to 70% [71, 76, 77]. This may be due to differences in assay sensitivity [71]. Farhath et al. found 92% pepsin positivity on analysis of tracheal aspirates from 45 preterm ventilated neonates, and correlated pepsin levels with development of bronchopulmonary dysplasia [78]. These studies, with their very high incidence of positive pepsin from lung secretions, along with other studies showing positive pepsin and bile acids in normal control subjects [79, 80] seem to suggest these assays may be overly sensitive [81]. Detection of milk protein by various methods has been utilized but has had very little replicative work [81-83]. These tests all suffer from lack of standardization and unknown effects of dilution from lavaging. The short half lives of these markers may make them applicable for detecting only recent aspiration events [81].

The most extensively studied biomarker for aspiration is the calculation of a quantitative index of lipid-laden macrophages (LLM) in bronchoalveolar lavage samples. Colombo and Halberg reported on the use of a LLM index in a group of children suspected to have aspiration during feeding. Using a 0 to 400 scale, they reported that children with suspected aspiration had a LLM index > 86 with the highest index in the children that were not aspirating being 72 [84]. Furuya et al. reported that a LLM index >165 had a 98% sensitivity, 78% specificity, and 87% overall accuracy as a diagnostic test for aspiration in a group of 112 children [85]. A LLM index >90 for children felt to be aspirating versus those not felt to be aspirating was retrospectively reviewed in 1999. The sensitivity and specificity of the test were 0.68 and 0.79, respectively [86]. The overall sensitivity and specificity of the LLM index to detect aspiration in infants and children as compared to a battery of other clinical and imaging exams is 69% to 98.6% and 78% to 79%, respectively [2, 84-86].

There have been subsequent studies that question the diagnostic utility of the LLM index in the evaluation of aspiration in children. In a retrospective chart study in a group of children with primary diagnoses of immunodeficiency, recurrent pneumonia, aspiration, trachea-bronchomalacia, and cystic fibrosis who had undergone bronchoscopy with bronchoalveolar lavage, the LLM index was determined. There was wide variability between the range of the LLM index and the primary diagnosis. The authors felt that the diagnostic utility of the LLM index is limited and should be interpreted with caution [87]. In another study a group of children with recurrent wheezy bronchitis and bronchial hyperreactivity or recurrent pneumonia with chronic cough underwent 24-hour pH monitoring and bronchoscopy with bronchoalveolar lavage. There were no correlations between pH-monitoring parameters and the counts of LLMs in the whole study population. The authors concluded that quantifying LLMs from bronchoalveolar lavage in children with gastroesophageal-related respiratory disorders does not have acceptable specificity to prove chronic aspiration as an underlying etiology [88]. The determination of the LLM index by inexperienced pathologists and cytotechnologists can be imprecise and can involve both inter and intraobserver bias [89, 90]. The LLM index may also vary depending on the amount of time since the last aspiration event [91]. Elevated LLM indexes have been found in children, not suspected of aspirating who have various diseases including cystic fibrosis, those receiving intravenous lipid preparations, those with pulmonary fat embolism in sickle cell disease, and those with endogenous lipoid pneumonia from bronchial obstruction [92-96]. Despite all of these limitations, a LLM index may provide supporting evidence of aspiration in select patients [97].

PULMONARY FUNCTION TESTS IN INFANTS WITH DYSPHAGIA

Sequelae of pulmonary aspiration associated with dysphagia in children can include infections such as pneumonias and pulmonary abscesses. Other children can also develop chronic conditions such as bronchiectasis, pulmonary fibrosis, or bronchiolitis obliterans. When these conditions occur, they require treatment and monitoring [2]. One of the tools now available for monitoring chronic respiratory problems caused by dysphagia and chronic aspiration in infants is pulmonary function tests (PFTs). Tutor et al. [98] reported on a group of 38 neurologically normal infants born at term, each with a history of recurrent cough and wheezing and who had dysphagia newly diagnosed using VFSS. These infants had PFTs performed within 2 weeks of their diagnosis of dysphagia. Twenty-five of them had abnormal spirometric values, 18 had abnormal plethysmographic values, and 15 demonstrated evidence of bronchodilator responsiveness (BDR). Seventeen of the 38 infants underwent repeat PFTs 6 months later after receiving therapy for their dysphagia. Ten of the infants continued to have abnormal spirometric values, two infants’ spirometric values remained normal, three infants’ abnormal spirometry had normalized, and two infants’ previously normal spirometric values had become abnormal. Eight of the 17 infants had continued abnormal plethysmographic values, six had continued normal plethysmographic values, and three infants’ previously normal plethysmographic values had become abnormal. Eight of the 17 infants demonstrated BDR, of which 5 continued to demonstrate BDR after 6 months of therapy for dysphagia and three infants developed BDR. The remainder continued to not be BDR (2 infants) or lost BDR (3 infants). The authors concluded that 6 months of therapy for dysphagia did not significantly improve pulmonary function in these infants. Future long-term studies will be necessary to determine if these PFT abnormalities persist into later childhood or even adulthood.

TREATMENT OF DYSPHAGIA

The approach to intervention for feeding and swallowing issues is individualized for each patient as they tend to be multifactorial in nature. Treatment approaches to feeding and swallowing issues for infants and children are composed of direct rehabilitative maneuvers/exercises and the use of compensatory strategies such as changes in positioning, changes/modifications to utensils (including bottle/nipple systems), modifications to the viscosity of the liquids (thickening liquids), and targeting improved swallowing function through exercises and maneuvers that compensate for poor swallowing function or help improve it [4, 43-45, 99]. Initial decisions for the treatment of dysphagia must take into account the prognosis for safe and adequate oral intake. The safest, most effective method of caloric intake may be orally with compensatory strategies or temporary/permanent feeding tube placement [43, 45].

In clinical practice, therapy intervention for children with oral-phase swallowing problems generally involves exercises aimed at improving the sensory and/or motor skills required for drinking and eating [100]. In a systematic review of the literature regarding the effect of oral motor exercises on swallowing in children, the authors concluded that there is insufficient evidence to determine the effects of these exercises on children with dysphagia [101]. Others have cautioned that these exercises should not be used as the only treatment for children with dysphagia and encouraged the use of functional therapy tasks that directly impact on eating and drinking ability and safety [102].

For children with swallowing problems affecting the pharyngeal phase, therapy intervention generally involves the child to modify their swallowing strategy or teaching the feeder to modify the bolus [100]. In a systematic review investigating the influence of thickening liquids on swallowing physiology and function, the authors concluded that thicker liquids reduce the risk of laryngeal penetration and aspiration but also increase the risk of post-swallow residue in the pharynx [103]. Treatment of gastroesophageal reflux disease, which can result in decreased laryngopharyngeal sensitivity, can lead to significant improvements in both swallow function and sensory testing [104].

In cases where a neurologically intact child, has dysphagia, many clinicians advocate that an assessment of the upper airway should be performed to assess for structural malformations. In addition, regardless of the etiology and treatment, it has been suggested that the return to a normal diet in children with dysphagia requires a gradual approach to allow systematic neuromuscular training of the pharyngeal phase of swallowing [105]. In another study in a group of children with chronic aspiration due to dysphagia but who had normal upper airway anatomy, the authors not only recommended feeding and swallowing therapy for the children, but they also recommended brain magnetic resonance imaging for patients with a suspected brainstem or posterior fossa lesion based on neurologic examination findings. They felt that most patients who aspirate thin and thickened liquids will have resolution of their swallowing dysfunction within 1 year of beginning therapy [106].

GASTROSTOMY/JEJUNOSTOMY TUBE PLACEMENT FOR SEVERE DYSPHAGIA

Children with dysphagia and chronic aspiration who continue to have significant recurrent respiratory symptoms despite the previously mentioned therapy methods may ultimately need the placement of a temporary or permanent feeding tube. This may become necessary acutely if there is inadequate intake of fluids or calories to prevent dehydration or malnutrition. The feeding tubes can be placed nasogastrically or nasojejunally but caregivers must be adequately trained in the proper insertion of nasogastric/nasojejunal tubes to prevent their migration into the lower airway with the significant risk of iatrogenic aspiration pneumonia [2, 107]. This approach is generally used only for short-term treatment [2]. The development of pyloric stenosis has been reported in three infants who received nasojejunal tube feedings for 3 to 4 weeks [108]. The incidence of gastroesophageal reflux is increased in children during transpyloric feedings [109] and in preterm infants, the incidence of gastrointestinal disturbances and mortality are increased in those who receive transpyloric feedings [110].

Surgical gastrostomy or jejunostomy tubes may be placed as an open procedure or by percutaneous or endoscopic methods [40]. Since it is more difficult to perform fundoplication after a gastrostomy, many surgeons prefer to have an evaluation done to exclude gastroesophageal reflux before placement of a gastrostomy tube [2].

Multiple problems can occur after placement of a gastrostomy tube. Major complications have been reported in 2% to 10.5% of children who have gastrostomy tube placement with the incidence of minor complications ranging from 16.4% to 73.6% [111-113]. The development of granulation tissue around the tube site was the most common problem sited in two studies occurring in 58% and 68% of patients, respectively [114-115] followed by tube dislodgement noted in 28% of patients in one of the studies [114]. The stoma may close quickly after the tube is accidently removed making reinsertion difficult if not impossible without surgical intervention. Other complications include new or deteriorating gastroesophageal reflux, infection, intra-abdominal leakage of food, and gastrocolic fistulae [116].

Both major and minor problems can occur with placement of a feeding jejunostomy [117-119]. In a report involving 89 patients who underwent placement of the jejunostomy, 15 patients (15.2%) had complications. Minor complications (7.2%) included dislodgement, blockage of the tube, and pericatheter leak. Major complications which required surgical intervention included detachment of the jejunostomy from the abdominal wall, leak into the peritoneal cavity, jejunal perforation by the catheter tip, and peritonitis after removal of the tube. Procedure related mortality was 3.2% [120]. Also if the tube is accidently removed, the stoma may close rapidly making tube reinsertion difficult, if not impossible, without surgical intervention.

The decision to place a feeding tube either on a temporary or permanent basis has long-reaching implications in the development of social, learning, and emotional skills [44-48].

Some children, particularly those with neurological impairment, may have an antireflux procedure, such as a fundoplication, performed at the time of placement of a gastrostomy tube. Several studies have reported worsening or de novo development of gastroesophageal reflux after placement of a gastrostomy tube alone in 5% to 67% of these children. Between 5% to 34% of children will require anti-reflux surgery such as fundoplication, to control their symptomatic gastroesophageal reflux [40, 121-122]. A recent study reported that Thal fundoplication in neurologically normal children with chronic airway diseases and documented gastroesophageal reflux disease resulted in significant improvement of their airway symptoms and a marked reduction in their need for medication [123].

Performance of a fundoplication can be associated with several serious complications such as liver laceration, bowel perforation, bleeding, peritonitis, “slipped” or disrupted fundoplication, dehiscence, hiatal hernia, and bowel obstruction [124-125]. It has been reported that 5% of Nissen fundoplications and 11.9% of Thal fundoplications required surgical revision [126-127]. Frequent long-term problems have included gas bloating, dumping syndrome, esophageal dysmotility, achalasia, inability to vomit or burp, slow eating, and dysphagia [126, 128]. Fundoplication can be associated with subsequent mortality in children. In a retrospective study of 412 children who underwent Nissen fundoplication, 15.3% subsequently died within a median of 6 months after the surgery. Factors associated with mortality in the study included surgical complications, neurological impairment, female gender, and fundoplication performed before the age of 18 months [129].

Despite placement of a fundoplication to reduce gastroesophageal reflux and placement of a surgical tube for feeding, some children with dysphagia will continue to have symptoms and signs of aspiration. At that point, one needs to determine if salivary aspiration is occurring, and if so, effective therapy needs to be provided [2].

MANAGEMENT OF SALIVARY ASPIRATION

Treatment of salivary aspiration may be medical or surgical. Anticholinergic agents, such as glycopyrrolate or scopolamine patches, have been used, especially in neurologically impaired children, to treat sialorrhea. Although effective in decreasing salivation, anticholinergic therapy can be associated with significant adverse side effects, including behavioral changes, constipation, dry mouth, thick secretions, urinary retention, constipation, flushing, nasal congestion, vomiting, diarrhea, and tachycardia. Treatment may need to be discontinued in up to one third of patients [40, 130-135].

Several studies have shown that injections of botulinum toxin, particularly type A, into the parotid or submandibular salivary glands with sonographic guidance, are effective in controlling sialorrhea in children, particularly those with neurological impairment [136-140]. Up to 88% of the patients have a decrease in saliva production [137]. Some researchers also reported a decrease in the number of hospitalizations and pulmonary infections after the injections, as well as, reduced pulmonary toilet requirements and reduced use of anticholinergic medication [138]. The reduction in saliva production ranged from 6 to 28 weeks in one study [139]. Reported complications have included self-limited oral bleeding, viscous saliva, parotitis, transient xerostomia, and dysphagia [136-137, 139-140]. There has been a report of the development of antibodies to botulinum toxin, resulting in no clinical response after treatment of sialorrhea with botulinum toxin [141]. In a study of 30 children with various neuromuscular disorders and dysphagia, the injection of botulinum toxin into the submandibular glands gave a reliable assessment of how much the salivary flow would be reduced after the submandibular gland had been removed [142]. In some centers, the use of botulinum toxin has been discontinued due to migration of the toxin from the injection site resulting in paralysis of critical bulbar functions resulting in acute deterioration and death in children and adults [2].

Surgical management of sialorrhea is an option in children for whom medical management fails. This often involves bilateral submandibular and parotid duct ligation or submandibular gland excision with parotid duct ligation. Studies have reported varying results, from up to an 87% success rate in controlling drooling [143-146] to minimal control of sialorrhea with surgery [147, 148]. In one small study of 12 children with neurological impairment, surgery was effective in reducing hospital admissions due to aspiration pneumonia [149].

Children who continue to aspirate and have recurrent pneumonias despite other medical/surgical therapies may need placement of a tracheostomy, particularly one using a cuffed tracheostomy tube, for pulmonary toilet. Despite this, there is lessened but still present risk of continued aspiration [2]. A recent study involving 4 toddlers with long-term tracheostomies revealed the presence of delayed swallow response initiation with associated penetration. Pharyngeal dysmotility was not observed [36]. The use of a tracheostomy speaking valve has been reported to reduce but not eliminate occurrences of aspiration in adults [37]. However, in a recent study, the presence of a speaking valve improved piriform sinus residue but did not decrease laryngeal aspiration or penetration in a group of children with tracheostomies [150].

The definitive treatment for the elimination of chronic pulmonary aspiration is laryngotracheal separation. This procedure eliminates all continuity between the respiratory and digestive tracts by disconnecting the upper trachea from the larynx and diverting it directly to a stoma. The proximal trachea is diverted to the esophagus which allows drainage of pooled secretions. Alternately, the larynx is simply closed, and oral secretions that accumulate in the larynx are either orally expressed or swallowed [2]. There is a loss of phonation, and the patient is left with a permanent tracheostomy [40]. This surgery, in several studies, has resulted in aspiration control and improved respiratory condition in between 63% to 100% of patients [151-155]. However, complications can be considerable, such as tracheal granulations, bleeding, stenosis of the tracheal stoma, tracheomalacia, tracheal abscess, ruptured sutures, tracheocutaneous fistula [157], and tracheoinnominate artery fistula [151, 154, 157]. Even if this procedure is performed without laryngectomy, laryngotracheal separation was felt to likely not be reversible [40]. However, the successful reversal of this surgery in 2 children has recently been reported [158].

CONCLUSION

Chronic aspiration can occur in children with dysphagia when there is failure of the airway protective reflexes to prevent it. There are several tests currently available to demonstrate the presence of dysphagia and pulmonary aspiration. Pulmonary function tests in infants demonstrate that significant impairment occurs in the lungs when there is chronic aspiration. Also, chronic aspiration in children with dysphagia can lead to the development of several pulmonary disease processes which can cause significant morbidity and possibly mortality for the patients. However, there are several effective medical and surgical procedures now available to treat infants and children with dysphagia to try to prevent or lessen the occurrence of pulmonary aspiration. These therapies have resulted in favorable outcomes in many children with dysphagia [159] along with improvement in their respiratory prognosis.

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BIOGRAPHICAL SKETCH

James Dudley Tutor, MD

Education:

High School:	Greenville High School
	Greenville, Mississippi
	Date of Graduation May 29, 1975
Undergraduate:	
January 1976 to	Delta State University
May 1979	Cleveland, Mississippi
	B. S. Degree May 1979
Medical School:	
July 1979 to	University of Mississippi School of Medicine
June 1983	Jackson, Mississippi
	M. D. Degree May 1983
Internship:	
July 1983: to	Internship - Pediatrics
June 1985	University of Mississippi School of Medicine
	Jackson, Mississippi
Residency:	
July 1985 to	Residency - Pediatrics
June 1987	University of Tennessee - Chattanooga Unit

	T. C. Thompson Children's Hospital Chattanooga, Tennessee
Postgraduate:	
July 1990 to June 1993	Fellowship - Pediatric Pulmonology Tulane University School of Medicine New Orleans, Louisiana
July 1992 to June 1993	Chief Fellow

Other Educational Training:

1991	Pediatric Flexible Bronchoscopy Instructional Course University of North Carolina at Chapel Hill

Honors:

1979	Beta Beta Beta Biological Honor Society
1979	Phi Kappa Phi Honor Society
1990	Who's Who in Mississippi (Medicine)
1993	Recipient of Second Place Research Award at Louisiana State University Medicine Research Day
1993	Recipient of American Federation of Clinical Research and Investigation Trainee Travel Award.
1998	Recipient of Bea Gerber Award from LeBonheur Club of Memphis
2011	Best Doctors in Memphis

Board Certification:

June 1988 - 1997	Diplomat of American Board of Pediatrics
August 1994 - Present	Pulmonology Sub-Board of American Board of Pediatrics

Licensure:

Tennessee	#MD16295	Issued June 12, 1985	Active
Kentucky	#29984	Issued September 19, 1993	Inactive
Mississippi	#10454	Issued July 1, 1984	Active
Louisiana	#08227R	Issued December 7, 1989	Inactive

Society Memberships:

1988-Present	Fellow, American Academy of Pediatrics and Section on Pulmonology
1991-Present	American Thoracic Society
1994-2001	Fellow, American College of Chest Physicians
1993-1995	Kentucky Medical Association
1993-1995	Kentucky Section of the American Academy of Pediatrics
1993-1995	Jefferson County Medical Society
1993-1995	Kentucky Commission for Handicapped Children
1994-1995	Louisville Pediatric Society
1995- 2007	Tennessee Section of the American Academy of Pediatrics
1996- Present	Christian Medical & Dental Associations
2011-Present	Memphis Medical Society
2011-Present	Tennessee Medical Association

University Appointments:

July 1993 to March 1995	Assistant Professor of Pediatrics Section of Pulmonology Department of Pediatrics University of Louisville School of Medicine Louisville, Kentucky
April 1 1995 to June 30, 2001	Assistant Professor of Pediatrics Section of Pulmonology Department of Pediatrics University of Tennessee-Memphis School of Medicine Memphis, Tennessee
July 1, 2001 to Present	Associate Professor of Pediatrics Section of Pulmonology Department of Pediatrics

	School of Medicine University of Tennessee-Memphis Memphis, Tennessee (Tenure awarded July 1, 2001.)
April 1995 to October 2005	Associate Medical Director Cystic Fibrosis Center University of Tennessee-Memphis Memphis, Tennessee
October 2005 to July 2007	Co-Medical Director Cystic Fibrosis Center University of Tennessee-Memphis Memphis, Tennessee
April 1995 to December 2008	Medical Director Pulmonary Function Laboratory LeBonheur Children's Medical Center Memphis, Tennessee
April 1995 to July 2007	Pulmonary Consultant Pediatric and Adolescent Sleep Disorder Center LeBonheur Children's Medical Center Memphis, Tennessee
October 1998 to December 2008	Associate Medical Director of Respiratory Care Services LeBonheur Children's Medical Center Memphis, Tennessee
April 1995 to Present	Medical Director Pulmonary Function Lab LeBonheur Children's Medical Center Memphis, TN
April 2009 to Present	Associate Director of Pediatric Pulmonology Fellowship Program

	University of Tennessee Health Science Center Memphis, Tennessee
November 1, 2011	Tenure voluntarily relinquished.
July 1, 2012 to Present	Professor of Pediatrics Section of Pulmonology Department of Pediatrics School of Medicine University of Tennessee Health Science Center Memphis, Tennessee

Hospital Appointments:

July 1983 to June 1985	University of Mississippi Medical Center Jackson, Mississippi - Active
July 1985 to June 1987	T. C. Thompson Children's Hospital Chattanooga, Tennessee - Active
July 1987 to June 1990	Kings Daughters Hospital Greenville, Mississippi - Active
July 1987 to June 1990	Delta Regional Medical Center Greenville, Mississippi - Active
July 1990 to June 1993	Tulane University Medical Center New Orleans, Louisiana - Active
July 1990 to June 1993	Medical Center of Louisiana New Orleans, Louisiana - Active
July 1993 to March 1995	Kosair Children's Hospital Louisville, Kentucky - Active
July 1993 to March 1995	Norton Hospital Louisville, Kentucky - Active

July 1993 to March 1995	Frazier Rehabilitation Center Louisville, Kentucky – Consultant
July 1993 to March 1995	Jewish Hospital Louisville, Kentucky - Consultant
July 1993 to March 1995	Audubon Regional Medical Center Louisville, Kentucky - Consultant
April 1995 to Present	Le Bonheur Children's Medical Center Memphis, Tennessee - Active
April 1995 to Present	The Med Memphis, Tennessee - Consultant
May 1995 to 1997	U. T. Bowld Hospital Memphis, Tennessee - Consultant
May 1995 to Present	St. Jude Children's Research Hospital Memphis, Tennessee - Consultant
June 1995 to 1999	Baptist Memorial Hospital Memphis, Tennessee - Consultant
September 1997 to Present	Methodist Hospitals of Memphis Memphis, Tennessee – Consultant
August 2008 to Present	North Mississippi Regional Medical Center Tupelo, MS – Consultant

Practice Experience:

July 1987 to June 1990	Partner in General Pediatric Private Practice Post Office Box 5216 1202 Hospital Street Greenville, Mississippi 38701
July 1993 to March 1995	Pediatric Pulmonary Medicine, P. S. C. Suite 201 233 East Gray Street

	Louisville, Kentucky 40202
April 1995 to December 2010	UT Medical Group, Inc.
January 2009 to Present	PSR, Inc. (Coverage at LeBonheur Childrens' Hospital ER and its associated Urgent Care Centers)
January 2011 to Present	UT Le Bonheur Pediatrics Specialists
January 2013 to Present	Children's Foundation Research Institute 50 North Dunlap Street Memphis, TN 38103

Teaching Experience:

July 1993 to March 1995	Lecture to 50 Medical Students 1 Hour Every 6 Weeks on Cystic Fibrosis
July 1993 to March 1995	Supervised 12 Medical Students and Pediatric Residents for One Month Pediatric Pulmonology Rotations
July 1993 to March 1995	Supervised Medical Students and Pediatric Residents Caring for Inpatients on Pediatric Pulmonology Service 26 Weeks/Year
April 1995 to Present	Supervise Medical Students and Pediatric Residents Caring for Outpatients in the Pediatric Pulmonology and Pediatric Cystic Fibrosis Clinics.

April 1995 to Present	Supervise Pediatric Residents for One Month Pediatric Pulmonology Rotation
April 1995 to July 2007	Supervise Medical Students and Pediatric Residents Caring for Inpatients on Pediatric Pulmonology Service for 26 Weeks per Year.
July 2007 to Present	Supervise Medical Students and Pediatric Residents Caring for Inpatients on Pediatric Pulmonology Service for 13 Weeks per Year.
April 1995 to December 1999	Lecture to 25 Medical Students 1 Hour Every 6 Weeks on Chronic Cough in Children
April 1995 to June 30, 2005	Supervise Pediatric Residents and Medical Students Caring for Inpatients on the General Pediatric Service for 1 Month/Year.
January 1996 – June 2007	Supervise Pediatric Residents and Fellows in Allergy/Immunology in Asthma Clinic 1 Afternoon/Six Weeks
July 2007 Present	Supervise Pediatric Residents and Fellows in Allergy/Immunology in Asthma Clinic 1 Afternoon/3 Months
February 26, 1996 to Present	Provide a Series of Lectures on Respiratory Physiology and Pulmonary Function Tests in Pediatric Patients to Fellows in Allergy/Immunology.

February 19, 1999	Lecture to pediatric Critical Care Physicians at LeBonheur Children’s Medical Center on Pulmonary Function Tests in Infants.
September 20, 2000, October 9, 200 February 23, 2005	Lecture to pediatric Allergy/Immunology Physicians at 2 LeBonheur Children’s Medical Center on Pulmonary Function Tests in Infants and Children.
July 2002 to Present	Supervise, as Attending, Pediatric Residents and Critical Care Fellows in the care of Pediatric Patients in the Transitional Care Unit for 1 Month/Year.
July 2005 to July 2007	Lecture to pediatric residents 26 times per year on various topics in Pediatric Pulmonology.
July 2007 to Present	Lecture to pediatric residents 13 times per year on various topics in Pediatric Pulmonology.
November 30, 2005	Lecture to pediatric Allergy/Immunology physicians at LeBonheur Children’s Medical Center on Aspiration in Infants and Children.
April 5, 2006	Lecture to pediatric Allergy/Immunology physicians at LeBonheur Children’s Medical Center on Interpretation of Pulmonary Function Tests.
May 28, 2008	Lecture to pediatric Allergy/Immunology Physicians at LeBonheur Children’s Medical Center on Evaluation of the Wheezing Infant.
June 17, 2008 to Present	Pediatric Pulmonology clinic in Tupelo, MS one day per month.

July 2008 to Present	Pediatric Pulmonary Journal Club presenter 1 hour, six times per year.
July 2011 to Present	Lecture at conference for pediatric pulmonary fellows and attendings.
July 2011 – June 2012	Faculty Mentor for four first year and three second year medical students at the University of Tennessee Health Science Center, Memphis, TN.
January 23, 2013	Lecture to 25 medical students on Wheezing in Infants.
April 4, 2014	Lecture to pediatric residents on Acute Infections Causing Upper Airway Obstruction
April 11, 2014	Lecture to pediatric residents on Dysphagia and Chronic Aspiration in Children.

Pulmonary Fellows Trained

Devon Greene, M.D.
Errin Newman, M.D.
Jaspal Hothi, M.D.
Andres Carrion, M.D.

Invited Lectures:

National:

1. Tutor JD. Emerging Technologies for Cystic Fibrosis. What's Pneu II Seminar for the Jefferson District of the Kentucky Society of Respiratory Care, Louisville, Kentucky, March 1994.
2. Tutor JD. Pediatric Bronchoscopy: Improved Tools, Expanding Uses. Owensboro Pediatric Society, Owensboro, Kentucky, May 1994.

3. Tutor JD. Pediatric Bronchoscopy: Improved Tools, Expanding Uses. Elizabethtown, Pediatric Society, Elizabethtown, Kentucky, September 1994.
4. Tutor JD. Mini-Internship: Discussion of Cases in Pediatric Pulmonary Medicine.
   Second Newborn and Pediatric Symposium. Kosair Children's Hospital in Conjunction with the University of Louisville School of Medicine, Louisville, Kentucky, October 14-15, 1994.
5. Tutor JD. Is Your Pediatric Patient Aspirating? North Mississippi Regional Medical Center, Tupelo, Mississippi, June 17, 2005.
6. Tutor JD. The Wheezing Infant. Lakeside Medical Center, Grenada, Mississippi. November 18, 2005.
7. Tutor JD. The Wheezing Infant. North Mississippi Regional Medical Center, Tupelo, Mississippi, April, 24, 2007.
8. Tutor, JD. Apnea of Prematurity, Acute Life-threatening Events, and Sudden Infant Death Syndrome. North Mississippi Regional Medical Center at Hamilton, Alabama, February 5, 2008.
9. Tutor, JD, Is Your Pediatric Patient Aspirating? Family Medical Faculty and Residents, Tupelo, Mississippi, October 21, 2008.
10. Tutor, JD. Is Your Pediatric Patient Aspirating? Pediatric Grand Rounds, Chattanooga, TN, January 7, 2009.
11. Tutor, JD. Evaluation and Management of Asthma in children. Tupelo, MS and New Albany, MS, February 26, 2016.

Local:

1. Tutor JD. Chronic Cough in Children. Eighteenth Annual Pediatric Symposium and Etteldorf Lectureship. LeBonheur Children's Medical Center in Conjunction with the University of Tennessee -Memphis College of Medicine, Memphis, Tennessee, March 15, 1996.
2. Tutor JD. Etiology of Cystic Fibrosis. Etiology and Management of Cystic Fibrosis.
   Pharma Thera, Inc. in Conjunction with the University of Tennessee - Memphis College of Pharmacy, Memphis, Tennessee, November 5, 1997.
3. Tutor JD. Complicated Parapneumonic Effusions and Empyema. Tennessee Society for Respiratory Care, Memphis, Tennessee, March 27, 1998.

4. Tutor, JD, Is Your Pediatric Patient Aspirating? Pediatric Nutrition Practice Group 3rd Quarter Meeting. LeBonheur Children's Medical Center, Memphis, Tennessee, August 12, 2008.
5. Tutor, JD Pediatric Grand Rounds, Pediatric Bronchoscopy. Le Bonheur Children's Medical Center, Memphis, TN, June 16, 2004.
6. Tutor, JD Pediatric Grand Rounds, Clinical Immunology Enrichment Conference PFTs/Infant PFTs. Le Bonheur Children's Medical Center, Memphis, TN, February 23, 2005.
7. Tutor, JD Pediatric Grand Rounds, Infant Wheezing. Le Bonheur Children's Medical Center, Memphis, TN, July 13, 2005.
8. Tutor, JD Pediatric Grand Rounds, New Recommendations for Management of Emphysema in Children. Le Bonheur Children's Medical Center, Memphis, TN, April 11, 2007.
9. Tutor, JD Pediatric Grand Rounds, Dysphagia and Chronic Aspiration. Le Bonheur Children's Medical Center, Memphis, TN, June 23, 2010.
10. Tutor, JD, Srinivasan, S. Pediatric Airway Conference. Airway Complications of Tuberculosis. LeBonheur Children's Hospital. July 22, 2011.
11. Tutor, JD._Pediatric Airway Conference. Pediatric Pulmonary Manifestations of Gastroesophageal Reflux, Dysphagia, and Aspiration. LeBonheur Children's Hospital. February 24, 2012.

Editorial Appointments:

Reviewer (JOURNALS):

Clinical Pediatrics -2001
Pediatric Pulmonology – 2008
PLOS ONE - 2015

Committees:

Hospital:

1993-1995	Kosair Children's Hospital Patient Care Committee
1993-1995	Kosair Children's Hospital Infection Control Committee
1999-7/2010	UT Medical Group, Inc. Credentialing Committee

2001-2002	Pulmonology Division Consultant to Credentials Committee at LeBonheur Children’ Medical Center
2008-2010	Asthma Community Committee at LeBonheur Children’s Medical Center
2008-Present	Cystic Fibrosis Quality Improvement Working Group
2008 –Present	Pediatric Pulmonology Fellowship Oversight Committee

University:

2005-2008	University of Tennessee Health Science Center-Committee on Academic Appointments and Promotions.
2012-Present	University of Tennessee Health Science Center-Admissions Sub-Committee.
2012-Present	University of Tennessee Health Science Center-Pediatric Department Promotion and Tenure Committee.

Other Activities:

June 12-18, 1994	Co-Medical Director, Camp Superkids, Memphis, TN
April 1996 to Present	Participate in Cystic Fibrosis Family Education Day on a yearly basis at Le Bonheur Children’s Medical Center, Memphis, TN.
May 4, 1996 to Present	Participate in Great Strides Walk-a-thon for the Cystic Fibrosis Foundation on a yearly basis in Memphis, TN.
January 1996 to Present	Member of Bellevue Baptist Church in Cordova, TN. Shepherd’s Ministry at Bellevue Baptist Church in Cordova, TN. Member of Benevolence Committee at

	Bellevue Baptist Church in Cordova, TN. Monitor in children's church one Sunday per month. Member of Contributions Committee Teacher of 6th grade boys Bible Fellowship class Teacher of 3 and 4 year old class one Sunday per month
March 1999 to May 2001	Secretary-Treasurer of Memphis chapter of the Christian Medical and Dental Society, Germantown, TN.
September 17 to September 23, 1999	Mission trip to Nicaragua sponsored by Bellevue Baptist Church, Cordova, TN.
May 13, 2000	Family Fun and Fitness Day at Bellevue Baptist Church in Cordova, TN.
September 14 to September 19, 2000	Mission trip to Guatemala sponsored by Bellevue Baptist Church, Cordova TN.
May 2001 to Present	Member of Continuing Education Committee of the Memphis chapter of the Christian Medical and Dental Associations. Memphis, TN.
November 10 to November 17, 2001	Mission trip to El Salvador sponsored by Bellevue Baptist Church, Cordova TN.
February 2005 to February 2006	Treasurer for Scout Committee at Heartsong Church Cordova, TN.
October 2006 to Present	Member, Academic Council of the Christian Medical and Dental Associations. Germantown, TN.

April 2007 to Present	Member of the Pediatrics section of the Christian Medical and Dental Associations, Germantown, TN.
June 22-29, 2007	Mission trip to Venezuela sponsored by Bellevue Baptist Church, Cordova, TN
July 2007 to May 2013	Member of the Leadership Council of the Memphis Chapter of the Christian Medical and Dental Associations, Memphis, TN.
anuary 2009 to Present	National Chairman, Academic Section of the Christian Medical and Dental Associations
April 12, 2012 April 3. 2014	Taught three comparative anatomy classes at Evangelical Christian School in Memphis, TN about the respiratory system and about the practice of pediatric pulmonology.
September 17, 2014 – September 28, 2014	Medical mission trip with the Medical Education International division of the Christian Medical and Dental Associations to Macedonia.

Research and Other External Support:

1993	Genentech, Inc., San Francisco, California: A Multi-Center Double-Blind Placebo-Controlled Protocol to Evaluate the Safety and Efficacy of Aerosolized rhDNase Therapy in Patients with Cystic Fibrosis Who Have Advanced Lung Disease. $9,000, (Co-Investigator)
1993	Genentech, Inc., San Francisco, California: A Multi-Center Epidemiologic Study to Evaluate Pulmonary Function and the Rate of Respiratory Tract Infections in Patients with Cystic Fibrosis. $12,500, (Co-Investigator).

1993	Cystic Fibrosis Foundation, Bethesda, Maryland. Cystic Fibrosis Center Grant. $24,890, (Co-Investigator, Louisville, KY)
1993	Genentech, Inc., San Francisco, California: A Multi-Center Open-Label Treatment Protocol to Provide Aerosolized rhDNase Therapy to Patients with Cystic Fibrosis. $1,250, (Co-Investigator.)
1994	Genentech, Inc., San Francisco, California: Epidemiologic Study of Cystic Fibrosis: A Multi-Center, Longitudinal Follow-up Study of Patients with Cystic Fibrosis for Monitoring Pulmonary Function, Pulmonary Exacerbations, and the Safety of Long-Term Treatment with Pulmozyme® (dornase alfa). $21,875, (Co-Investigator, Louisville, KY).
1994	University Pediatrics Foundation, Louisville, Kentucky: A Study of the Occurrence of Electrical Periodicity in Pediatric Patients with Status Asthmaticus. $1,149, (Principal Investigator).
1994	Burroughs Wellcome Co., Research Triangle Park, North Carolina: A Randomized, Placebo-Controlled Dose Ranging Study of Exosurf® Aerosol in Patients with Cystic Fibrosis Lung Disease. $22,000, (Co-Investigator).
1995	Cystic Fibrosis Foundation, Bethesda, MD. Cystic Fibrosis Center Grant. $35,000, (Co-Investigator, Memphis, TN).
1995-2005	Genentech, Inc., San Francisco, CA: Epidemiologic Study of Cystic Fibrosis: A Multi-Center, Longitudinal Follow-up Study of Patients With Cystic Fibrosis for Monitoring Pulmonary Function, Pulmonary Exacerbations, and the Safety of Long-Term Treatment with Pulmozyme® (dornase alfa), (Co-Investigator, Memphis, TN).
1995	Univax Biologics, Inc., Rockville, Maryland: A Randomized Multi-Center, Double-Blind, Placebo-Controlled Study to

	Evaluate the Efficacy of a Mucoid Exopolysaccharide *Pseudomonas aeruginosa* Immune Globulin Intravenous (Human) (MEP IGIV) in Reducing the Frequency of Acute Pulmonary Exacerbations in Patients with Cystic Fibrosis. $63,312, (Co-Investigator).
1995	Cystic Fibrosis Foundation, Bethesda, MD. Multicenter Epidemiologic Registry of Cystic Fibrosis (CF): A Longitudinal Follow-up Study of Patients with Cystic Fibrosis, for Monitoring Trends in Treatments, Patient Outcomes and Improvements in Patient Care. (Co-Investigator).
1997	UT Medical Group, Inc., Memphis, Tennessee: Methacholine Responsiveness in Infants, $8569, (Principal Investigator).
1997	Evaluation of the Efficacy, Safety, Tolerance and Pharmacokinetics of Meropenem for the Treatment of Acute Pulmonary Exacerbations in Patients with Cystic Fibrosis., $10,000, (Co-Investigator).
1998	LeBonheur Club, Memphis, Tennessee: Bea Gerber Research Award, $5,000, (Recipient).
1999	A Phase IV Study of the Safety of Synagis® (Palivizumab) for Prophylaxis of Respiratory Syncytial Virus Disease in Children With Cystic Fibrosis, (Co-Investigator).
1999	Member of Data and Safety Monitoring Group for A Phase II Study of Dexamethasone in Intubated Infants and Children with Respiratory Syncytial Virus Infection. Principal investigator: John Devincenzo, Memphis, Tennessee.
May 14, 2003	National Airway Clearance Registry (NACR) Program for Cystic March 31, 2005 Fibrosis, (Co-Investigator).

2006-2009	The EPIC Observational Study: Longitudinal Assessment of Risk factors for and impact of *Pseudomonas aeruginosa* Acquisition and early anti-Pseudomonal treatment in children (EPIC -002). Cystic Fibrosis Foundation. (Co-Investigator).
2006-Present	Effectiveness and safety of intermittent antimicrobial therapy for the treatment of new onset *Pseudomonas aeruginosa* Airway infection in young patients with cystic fibrosis (EPIC-001). Cystic Fibrosis Foundation. (Co-Investigator).
2007-Present	Genetic modifier in cystic fibrosis liver disease, University of North Carolina and the Cystic Fibrosis Foundation. (Co-Investigator).
2007-2009	An open-label clinical study evaluating the long-term safety of ALTU-135 for the treatment of patients with cystic fibrosis-related exocrine pancreatic insufficiency. Altus Pharmaceutical, Inc. (Co-Investigator).
2007-2009	A phase III, randomized, double-blind, placebo-controlled clinical study evaluating the efficacy and safety of ALTU-135 treatment in patients with cystic fibrosis-related exocrine pancreatic insufficiency Altus Pharmaceutical, Inc. (Co-Investigator).
2008-2009	Multi-center, multi-national, randomized, placebo-controlled trial azithromycin in subjects with cystic fibrosis 6-18 years old, and culture negative for *Pseudomonas aeruginosa*. Cystic Fibrosis Foundation. (Co-Investigator).
2008-2009	A Phase 2, Multi-center Randomized, Double-blind, Placebo-Controlled Study to Evaluate the Safety, Tolerability and Efficacy Of Three-Dosage Regimens of MP-376 Solution for Inhalation Given For 28 Days to Stable Cystic Fibrosis Patients. (Co-Investigator).

2008-2010	The Use of Formula Fortified with Docosahexaenoic acid (DHA) in Infants with Cystic Fibrosis. (Memphis CF Center PI).
7/1/08-6/30/10	A Pilot Study of Pulmonary Function in Dysphagic Infants. LeBonheur Foundation Small Grant. $4,000. (Principal Investigator).
2008-2011	Long Term Administration of Inhaled Dry Powder Mannitol in Cystic Fibrosis - A Safety and Efficacy Study. (Co-Investigator).
2008-2011	Genetic modifiers of cystic fibrosis-twins and siblings. John Hopkins Medical Institutions with sponsorship from the National Institutes of Health and the Cystic Fibrosis Foundation. (Co-Investigator).
8/2009-12/2009	Evaluation of Immunogenetics Kit for the Detection of Cystic Fibrosis Protein. (Co-Investigator).
2009-2011	Expanded Access Program for Aztreonam Lysine for Inhalation in Patients with Cystic Fibrosis and *Pseudomonas aeruginosa* Airway Infection Who Have Limited Treatment Options and are at Risk for Disease Progression. (Co-Investigator).
2009-2010	Immunogenicity and Safety of Inactivated H1N1 Swine-origin Influenza Monovalent Vaccine in Children and Young Adults with Cystic Fibrosis. (Co-Investigator).
2009-Present	A Phase 2, Randomized, Double-blind, Placebo-controlled, Parallel-group Study to Evaluate the safety and Efficacy of VX-770 in Subjects Aged 12 Years and Older with Cystic Fibrosis Who Are Homozygous for the F508del-CFTR Mutation. (Co-Investigator).

2010-Present	A Phase 3 Multi-center, Multi-national, Randomized, Double-blind, Placebo-controlled Study to Evaluate the Efficacy and Safety of MP-376 (Levofloxacin Inhalation Solution; Aeroquin®) in Stable Cystic Fibrosis Patients. (Co-Investigator).
2010	Research Survey: the 2009-2010 H1N1 Influenza A Experience in U.S. Cystic Fibrosis Centers. (Co-Investigator).
4/2010-2011	Assessing Glutamine Blood Levels and Resting Energy Expenditure in the Pediatric Population. (Principal Investigator).
2011-Present	A Phase 3, Open-Label Randomized Trial to Evaluate the Safety and Efficacy of MP-376 Inhalation Solution (Aeroquin®) Versus Tobramycin Inhalation Solution in Stable Cystic Fibrosis Patients. (Co-Investigator).
2011-Present	A Cell Phone Support Program for Adolescents with Cystic Fibrosis. (Co-Investigator).
2012-Present	A 10 year Prospective Observational Study of the Incidence of and Risk Factors for Fibrosing Colonopathy in US patients with Cystic Fibrosis. (Co-Investigator).
6/14/12-6/5/13	A Retrospective Study of Pulmonary Function in Infants Diagnosed with Dysphagia. (Principal Investigator).
2013-Present	A Phase 3, Randomized Double-Blind, Placebo-Controlled, Multicenter Study of Aztreonam for Inhalation Solution (AZLI) in a Continuous Alternating Therapy (CAT) Regimen of Inhaled Antibiotics for the

	Treatment of Chronic Pulmonary Pseudomonas aeruginosa Infection in Subjects with Cystic Fibrosis. (Co-Investigator).
2013-Present	A Phase 2, Randomized, Double-blind, Placebo-controlled, Repeat-dose Study of KB001-A in Subjects with Cystic Fibrosis Infected with Pseudomonas aeruginosa. (Co-Investigator).
2013-Present	A Phase 3, Randomized, Double-Blind, Placebo-Controlled, Parallel-Group Study to Evaluate the Efficacy and Safety of Lumacaftor in Combination with Ivacaftor in Subjects Aged 12 Years and Older with Cystic Fibrosis, Homozygous for the F508del-CFTR Mutation. (Co-Investigator.)
2013-Present	OPTIMIZing Treatment for Early Pseudomonas aeruginosa in Cystic Fibrosis: The OPTIMIZE Multicenter, Placebo-Controlled, Randomized Trial. (Co-Investigator).
2014-Present	A Phase 3, Rollover Study to Evaluate the Safety and Efficacy of Long-term Treatment with Lumacaftor in Combination with Ivacaftor in Subjects Aged 12 Years and Older with Cystic Fibrosis, Homozygous or Heterozygous for the F508del-CFTR Mutation. (C0-Investigator).
2014-Present	The EPIC Observational Study: Longitudinal Assessment of Risk Factors for and Impact of Pseudomonas aeruginosa Acquisition and Early Anti-Pseudomonal Treatment in Children with CF. (Co-Investigator).
2014	A Phase 2, Randomized, Multicenter, Double-Blind, Placebo-Controlled Study to Evaluate Safety, Efficacay, Pharmokinetics, and Pharmodynamics of

VX661 in Combination with Ivacaftor for 12 Weeks in Subjects with Cystic Fibrosis, Homozygous for F508del CFTR Mutation. (Co-Investigator).

Publications (Book Chapters)

1. Tutor, JD, Haberman B. Respiratory and sleep disorders in children with myotonic Dystrophy. In: Myotonic Dystrophies: Epidemiology, Diagnosis, and Therapeutic Challenges. Sandra Jenkins, ed. Hauppauge, NY; Nova Science Publishers, Inc. 2015.
2. Tutor, JD. Respiratory problems in children with dysphagia and chronic aspiration. In: Gosa, MM, Suiter D, eds. Dysphagia: diagnosis and Treatment in Children and Adults. New York, NY; Thieme. (Book to be published in 2016. Chapter submitted on June 15, 2015.)

Publications (Journals):

1. Tutor JD. Therapy Update: I.T.P. of Childhood. Southeast Pediatrics 1986; Spring: 3-7.
2. Tutor JD, Kiernan MP, Beckerman RC. Pediatric Bronchoscopy: Improved Tools, Expanding Uses. Journal of Respiratory Diseases 1991;12:1016-35.
3. Tutor JD, Beckerman RC, Davis PW. The Teenager with Cystic Fibrosis: Overcoming the Psychosocial Hurdles of Adolescence. Newsletter of the Section of Adolescent Health of the American Academy of Pediatrics 1992;13:11-19.
4. Tutor JD, Mason CM, Dobard E, Beckerman RC, Summer WR, Nelson S. Loss of Compartmentalization of Alveolar Tumor Necrosis Factor After Lung Injury. American Journal of Respiratory and Critical Care Medicine 1994;149:1107-11.
5. Tutor JD, Eid NS. Treatment of Idiopathic Pulmonary Hemosiderosis with Inhaled Flunisolide. Southern Medical Journal 1995;88:984-86.
6. Tutor JD, Eid NS. Pregnancy in Women with Pulmonary Fibrosis: A Case Report of Pregnancy in a Woman with Idiopathic Pulmonary Hemosiderosis. Adolescent and Pediatric Gynecology 1995;8:128-131.
7. Tutor JD, Montgomery VL, Eid NS. A Case of Influenza Virus Bronchiolitis Complicated by Pneumomediastinum and Subcutaneous Emphysema. Pediatric Pulmonology 1995;19:393-95.

8. Tutor JD, Schoumacher RA. Wheezing in Infants: Working Efficiently to the Cause. Journal of Respiratory Diseases 1996;17:977-97.
9. Tutor JD, Schoumacher RA. Wheezing in infants: Pursuing the ‘less obvious.’ Journal of Respiratory Diseases 1996;17:1039-46.
10. Tutor JD, Schoumacher RA, Fitch SJ. What caused the cough and chest pain in this 6-year-old girl? Journal of Respiratory Diseases 1999;20:45-49.
11. Tutor JD, Schoumacher RA, Chesney PJ. Chylothorax associated with histoplasmosis in a child. The Pediatric Infectious Disease Journal 2000;19:262-263.
12. Tutor JD. Dysphagia and aspiration in the pediatric patient. Special Interest Division 13 newsletter of American Speech-Language-Hearing Association, 2001, 10;2:6-9.
13. Tutor JD, Schoumacher RA. Is aspiration causing your patient’s symptoms? The Journal of Respiratory Diseases for Pediatricians 2001;3:132-138.
14. Tutor JD, Schoumacher RA. Is aspiration causing your patient’s symptoms? The Journal of Respiratory Diseases 2003:24;30-32, 37-40.
15. Tutor JD. How to spot parasitic diseases involving the lungs: part 1. The Journal of Respiratory Diseases. 2004:25;322-326.
16. Tutor JD. How to spot parasitic diseases involving the lungs: part 2. The Journal of Respiratory Diseases. 2004:25;356-361.
17. Tutor JD. Pertussis: A cause of cough in adults as well as children. The Journal of Respiratory Diseases. 2008:29;172-178.
18. Tutor JD, Gosa MM. State of the art: Dysphagia and aspiration in children. Pediatric Pulmonology. 2012:47:321-337.
19. Tutor JD. Chylothorax in infants and children. Pediatrics. 2014;133:722-733.
20. Tutor JD, Srinivasan S, Gosa MM, Spentzas T, Stokes DC. Pulmonary function in infants with swallowing dysfunction. PLOS ONE. Published May 15, 2015. DOI: 10.1371/journal.pone.0123125.

Publications (Abstracts):

1. Tutor JD, Dyson MC, Beckerman RC, Osgood T, Kadowitz PJ. Comparison of Airway and Pressor Responses to Endothelin Peptides in the Cat. American Review of Respiratory Disease 1991;143:A159.

1. Poster Presentation: International Conference of the American Lung Association/American Thoracic Society, May 12-15, 1991 at Anaheim, California.

2. Poster Presentation: 1991 Annual Meeting of the American Academy of Pediatrics, October 26-31,1991 at New Orleans, Louisiana.
Pediatrics, October 26-31, 1991 at New Orleans, Louisiana

2. Tutor JD, Beckerman RC, Pickoff A, El-Dahr J. Occurrence of Electrical Periodicity in Pediatric Patients with Status Asthmaticus. Journal of Allergy and Clinical Immunology 1993;91:318.

1. Oral Presentation: Annual Conference of the American Academy of Allergy and Immunology, March 12-17, 1993 at Chicago, Illinois.

3. Tutor JD, Mason C, Beckerman RC, Summer W, Nelson S: Lung Injury isAssociated with Loss of Compartmentalization of Alveolar Tumor Necrosis Factor in the Isolated Perfused Rat Lung. American Review of Respiratory Disease 1993;147:A730.

1. Oral Presentation: Louisiana State University Medicine Research Day, January 15, 1993 at New Orleans, Louisiana.
2. Oral Presentation: Annual Meeting of the Southern Section of the American Federation of Clinical Research and Investigation, January 21-23, 1993 at New Orleans, Louisiana.
3. Oral Presentation: Louisiana State University Cardiovascular Medicine Research Day, May 13, 1993 at New Orleans, Louisiana.
4. Poster Presentation: International Conference of the American Lung Association/American Thoracic Society, May 16-19, 1993 at San Francisco, California.

4. Tutor JD, Eid NS. Treatment of Idiopathic Pulmonary Hemosiderosis with Inhaled Flunisolide. Southern Medical Journal 1995;88:984-986.

1. Oral Presentation: Annual Meeting of the Kentucky Thoracic Society, March 25, 1994 at Lexington, Kentucky.
2. Poster Presentation: 1st International Congress on Pediatric Pulmonology, June 2-5, 1994 at Nice, France.

5. Eid NS, Eddy M, Tutor J, Franco S, Yandell B, Colliver L. Cost-Containment in Childhood Asthma.

   1. Oral Presentation: American Academy of Allergy and Immunology/ American Thoracic Society Conference: “Asthma 1995: Theory to Treatment,” July 15-17, 1995 at Chicago, Illinois.

6. Tutor JD, Srinivasan S, Gosa M, Culbreath B, Mallory L, Redd L, Jones M, Curry R, Davis S, Christensen, M, Schoumacher R, and Stokes D. Pulmonary Function in Infants with Chronic Aspiration. American Journal of Respiratory and Critical Care Medicine 2010;181:A6229.

   1. Poster Presentation: International Conference of the American Thoracic Society, May 14-19, 2010 at New Orleans, Louisiana.
   2. Poster Presentation: 3rd Annual Pediatric Research Day at LeBonheur Childrens Hospital, Memphis, Tennessee, November 17, 2010.

7. Stokes DC, Ward JC, Ferguson J, Rago D, McKeever CD, Srinivasan S, Schoumacher R, Tutor JD, Lee S. Cystic fibrosis in a Premature Native African Infant Diagnosed by Newborn Screening: Challenges in Diagnosis and Management. American Journal of Respiratory and Critical Care Medicine 2010;181:

   1. Poster Presentation: International Conference of the American Thoracic Society, May 14-19, 2010 at New Orleans, Louisiana.

8. Srinivasan S, Schoumacher RA, Tutor JD, Stokes DC. Pulmonary Alveolar Proteinosis, Piebaldism, and c-kit Mutation.

   1. Poster Presentation: Diffuse Lung Disease in Children 2012. June 14-15, 2012 at San Diego, California.

Publications (Non-Journal)

Interviews

1. Tutor, JD. Interviewed for article: Breathing Easy. Tupelo Daily Journal. March 27, 2009.

2. Tutor, JD. Interviewed for article: Take a Deep Breath. Living Well with Asthma. Vim & Vigor. Summer 2010.

Letters to the editor (Memphis Commercial Appeal)

1. Tutor, JD. Blackburn Failed Us on Bailout, October 6, 2008.
2. Tutor, JD. God’s Wrath Arrives: Obama, January 30, 2009
3. Tutor, JD. Embryonic Stem Cell Research, March 22, 2009.
4. Tutor, JD. A Divine Answer on Stem Cells, September 15, 2010.
5. Tutor, JD. Big Paydays Drive Tort-Reform Foes, March 8, 2011.
6. Tutor, JD. Fear of the Needle Puts Kids at Risk, December 4, 2011.
7. Tutor, JD. Illegal Tax Levied for Secret “Citizenship,” September 10, 2012.
8. Tutor, JD. The Bible says…, October 19, 2012.
9. Tutor, JD. That Carter Legacy, October 24, 2013.
10. Tutor, JD. Vaccines Save Lives, May 17, 2014.

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Chapter 2

POST-STROKE DYSPHAGIA

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"Sapienza" University of Rome, Rome, Italy

ABSTRACT

Dysphagia is a common consequence after stroke, presenting in up to 50% of all acute stroke patients, with increased morbidity and mortality mainly because it dramatically increases the risk of aspiration pneumonia, and because it leads to malnutrition and dehydration as well.

It has been reported that the incidence of dysphagia ranges from 25% to 81% depending on the timing of the assessment, diagnostic methods and criteria.

The nervous system affected by a stroke damage is thought to reserve a variable ability to somehow reorganize damaged pathways and structures, allowing an effective recovery from swallowing impairment.

Notwithstanding, about half of stroke patients remain dysphagic after seven days from symptoms onset, and approximately 13-15% of patients has persistent swallowing dysfunction after six months, with a dramatic impact on patients' outcome in terms of increased likelihood of residential placement and estimated lifetime costs in stroke survivor.

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To date, it is unclear why some stroke patients develop a persistent swallowing impairment after stroke or, in other words, if the failure in recovery from dysphagia is due to a stroke involving the dominant swallowing hemisphere, rather than a swallowing key region, or even to any specific clinical or neuroradiological stroke factor.

In this view, stroke severity upon admission, assessed using NIHSS, is an important clinical predictor of post-stroke dysphagia and of a persistent pattern of dysphagia as well.

Regarding the site of the stroke lesion, it is widely accepted that swallowing function is provided by a complex neural network bilaterally involving the perirolandic sensorimotor cortex which, together with other cortical structures, projects and receives afferences to the brainstem' Central Pattern Generator, through the basal ganglia, the thalamus and the periventricular white matter as well. Despite several cortical areas have been described as related with swallowing impairment, a definite role in causing dysphagia has been not completely understood for all of them so far.

Interestingly, recent studies reported that the disruption of cortical-cortical and cortical-subcortical white matter connections seems to increase the risk of dysphagia and aspiration pneumonia by lowering the threshold of input to the medullary swallowing center.

Thus, dysphagia may result from the combined effects of an acute focal damage and a preexisting impairment of the subcortical white matter connecting pathways. In other words, we might think of dysphagia as a kind of deficit in executive functions, which we know to be particularly associated with leukoaraiosis and cognitive disorders, and of course age.

Keywords: leukoaraiosis, dysphagia, stroke

INTRODUCTION

Dysphagia is a common consequence after stroke, presenting in up to 50% of all acute stroke patients. It has been reported that the incidence of dysphagia ranges from 25% to 81% depending on the timing of the assessment, diagnostic methods and criteria.

The clinical and scientific relevance of post-stroke dysphagia is demonstrated by its high incidence and negative effect on clinical outcome, with increased morbidity and mortality mainly because it dramatically increases the risk of aspiration and pneumonia [1, 3-6].

In this view, pulmonary infections represent one of the most common and fearsome complications in stroke patients: SPREAD guidelines report that, along with urinary infections, it reaches a cumulative incidence of about 30%, hence far exceeding that of other internal medicine complications [7].

Aspiration pneumonia is also associated with a relevant worsening of stroke outcome: a recent multicenter observational study, carried out on 8,250 stroke patients, confirmed that lung infections in patients with stroke are associated with worsening of mortality both at 30 days and at one year after the acute phase of stroke, with an increased length of stay, a greater disability at discharge, and an increased percentage of stroke patients sent in long-term care [4].

In the literature there are also conflicting reports on the consequences of the so called "silent aspiration," which is the aspiration of swallowed material without any clinical manifestations, and thereby underdiagnosed. It is estimated to be prevalent in 15-39% of patients with subacute stroke and in 2-25% of unselected patients with acute stroke, with a relevant increase in relative risk of pneumonia and a poorer clinical outcomes for silently aspirating patients compared with overt/non-aspirators [8, 9].

This data, as well as the strong evidence that dysphagia significantly contributes to malnutrition and dehydration regardless of aspiration [10], highlight the dysphagia's dramatic impact on patients outcome, with increased likelihood of residential placement and significantly increases estimated lifetime costs in stroke survivors [11].

NEURAL CONTROL OF SWALLOWING

It is now widely recognized that swallowing process is mediated by neural pathways that involve cortical input reaching the brainstem by means of fibers, such as the corticobulbar tract, which connect cortical areas that control swallowing with bulbar nuclei that mediate the final mechanism; however, the role of the different cortical and subcortical areas in causing dysphagia is still not entirely clear.

Several studies have dealt with identifying those structures involved in the process of swallowing.

In the past, researchers have mainly focused their attention on specific regions of the brainstem, in order to evaluate the action of interneurons involved into the oral and pharyngeal swallowing.

The focus then shifted on the interaction between different cortical areas and brainstem nuclei to control swallowing [12-15].

At a subcortical level, a consistent peristaltic contraction is guaranteed by a number of interneurons located in the reticular formation, in the nucleus of the solitary tract and in the nucleus ambiguus as well, all regions which constitute the swallowing core regulator of the brainstem. These centers are responsible for the integration of information coming from the oropharynx, esophagus and from supranuclear structures and are classically described as "central pattern generator" (CPG).

CPG is mainly entrusted with the control of both timing and synchronism of swallowing process. In any case, the brainstem is a part of a complex cortical-subcortical network which involves a sensory system, motor system, and white matter pathways. In this point of view, the majority of the recent studies highlight that the integrity of these connections appears to be fundamental for an effective coordination of swallowing to be achieved [16-18].

Brainstem and Peripheral Control

Since early researches carried on by Car and coworkers [19, 20], the brainstem has been described as responsible for the complex sequences and execution of the neuromuscular events involved in swallowing, by means of the compact clustering of bilateral cranial nerves nuclei and interneuronal connections as well [21]. The very first evidence of this role came from a study of decerebrated animals [22]: it has been demonstrated an elicitation of pharyngeal and esophageal swallowing obtained by stimulating this area in the absence of cortical input.

It is now widely accepted that brainstem structures provide the basic motor plan for pharyngeal swallowing, being also involved in the coordinative interactions between swallowing and respiration [23].

As mentioned above, the swallowing core regulator of the brainstem is represented by the Central Pattern Generator (CPG), a structure located into the rostral and ventrolateral portion of the brainstem. From an anatomical and functional point of view, the CPG has been classically divided in two bilateral region [24]: a dorsal region, which includes the nucleus of the solitary tract (NTS) with the adjacent reticular formation, and a ventral region, represented by the area surrounding and including the nucleus ambiguus (NA).

Since NTS represents the primary sensory nucleus for the VII, IX and X cranial nerve, the dorsal region is thought to represent the initial neuronal entrance for input that modulates swallowing: sensitive afferent fibers originate from mucosal chemoreceptors, mechanoreceptors and thermoreceptors of the oral cavity, pharynx and larynx; these fibers are involved in both the initiation and repeated activation of pharyngeal phase of swallowing, and are essential for the protection of the airway by the phenomenon of aspiration.

Muscle spindle receptors located in the pharyngeal and esophageal swallowing muscle, are otherwise considered as a trigger for those interneurons that modify motor output to muscle [21].

Otherwise, CPR ventral region is involved in sending out neural commands that control pharyngeal, laryngeal, and esophageal muscles. This motor task is carried out by means of NA, which is the primary motor nucleus for IX, X and XI cranial nerves, and has several interconnections with other medullary motor nuclei, such as those of V, VII and XII cranial nerves.

NA also contains axons projected to contralateral brainstem regions, suggesting that the ventral region has also a role in the integration of sensory information; this suggestion is otherwise confirmed by the fact that the ventral efferent medullary region receives direct input from the dorsal afferent medullary one. Hence, it could be argued that sensory information entering the brainstem via the NTS is integrated by the NA into a swallowing motor plan, and then transmitted to the motor nucleus for execution [21].

Finally, the interneuronal networks localized in CPG integrates sensory information by modulating afferent and efferent pathways activation which reciprocally connects the brainstem to the higher centers. This circuit is thought to represent the way how motor circuits are modulated by sensory feedback pathways, depending on bolus characteristics, head position and on muscular activity.

Higher Nervous System Control

While the basic motor plan for pharyngeal swallowing is provided by brainstem structures, the start of swallowing is a voluntary act which depends on the integrity of several cortical areas.

It has been suggested that the programming of the swallowing process firstly occurs in non-primary motor areas (i.e., mesial premotor cortex), secondly involving the primary sensorimotor cortex, and finally reaching the secondary areas.

Several studies [25-29] have identified numerous areas of interest who have a significant role in swallowing, such as the premotor cortex, the primary sensorimotor cortex, the insula, as well as some limbic areas. However, although these foci have been consistently identified in many of these studies, the exact width and functional relevance of the entire swallowing network have not been entirely clarified so far.

Discrepancies between the studies probably reflect methodological differences: the main imaging techniques used to investigate the neural basis of swallowing (i.e., functional Magnetic Resonance Imaging (fMRI), Positron Emission Tomography (PET) and Magnetoencephalography (MEG)) actually offer a representation of different aspects of brain activity. fMRI detects changes in local cerebral oxygenation, closely linked to the underlying neuronal activity; PET uses radioactive markers to study local changes in neurophysiological parameters as cerebral perfusion and glucose consumption; finally MEG measure small magnetic fields arising from neural.

Among these, fMRI is the most widely adopted technique, mainly because of its safety and its spatial resolution of about 4 mm; hence according to some Authors it could be used as an auxiliary technique to the conventional MRI to investigate the dysphagia following brain injuries. However even fMRI studies are heterogeneous with regard to the task investigated (voluntary or reflex swallowing, number of swallows, etc.).

An interesting meta-analysis [30] based on the activation likelihood estimation (ALE), has compared some of these studies in order to better identify brain areas involved in both water and saliva voluntary swallowing. The ALE is a quantitative voxel-wise meta-analysis technique which estimates the probability of brain activation in a given voxel, by analyzing the results of several studies expressed as standard space coordinates of activation maxima. The meta-analysis of brain activation showed that the significant activation clusters seen during water swallowing were partly different from those seen during saliva swallow. Authors also performed a between condition meta-analysis showing that, despite a partial overlap in the network involved in water and saliva swallowing, the first is strongly associated with right inferior parietal activation, while the latter more strongly involves premotor areas. This datum suggests that water swallowing strongly activates those cortical areas involved in sensory processing of intraoral water stimulation, while saliva swallowing massively involves premotor areas, which are crucial for initiation and control of swallowing sequence movements.

To have an overview of the several cortical and subcortical regions involved in swallowing, Table 1 report those dysphagia regions of interest, as identified by a recent meta-analysis [31], describing both those areas for which there is a clear implication, identified by most studies, both those ones whose role is still under discussion. The hypothesized role in swallowing mechanisms is labelled as well.

Table 1. Cortical and subcortical regions involved in swallowing

Region of Interest (ROI) Selected For Analysis and Hypothesized Role in Swallowing		
ROI	Role	References
Primary Somatosensory, Motor and Motor Supplementary cortices (BA 1, 2, 3, 4, and 6)	Cortical processing of swallowing, including motor regulation and execution and sensorimotor control	Hamdy et al., Martin et al., Mosier and Bereznaya
Anterior cingulate (BA 24 and 32)	Higher order motor processing: swallowing movement planning and execution	Hamdy et al.
	Cognitive perceptual processes (attention and response selection)	Martin et al.
Orbitofrontal cortex (BA 10, 11, 12, 44, 45, 47)	Unclear	Mosier et al.
Parieto-Occipital cortex (BA 7,17, 18, 40)	Sensory processing of swallowing	Hadmi et al., Kern et al.
	Task-cue processing (not swallowing per se)	Toogood et al.
	Movement planning and execution	Mosier and Bereznaya
Insular Cortex	Processing of gustatory input	Daniels et al., Daniels and Foundas
	Intraoral sensory modulation	Mosier et al.
Internal Capsule	Functional connection of the cortical and brainstem nuclei via the corticobulbar tracts	Mosier et al.
Thalamus	Sensory and motor input processing via thalamocortical and thalamostriatal pathways	Daniel et al., Mosier et al.
Basal Ganglia (caudate and/or putamen)	Gating of Sensory Input	Mosier and Bereznaya, Daniel et al., Suzuki et al.
Cerebral Peduncle	Descending pathways from the cortex	Miller
Brainstem	Central pattern generator, swallowing regulation	Jean
Cerebellum	Regulation of adaptive coordination, sequencing, timing, learning and memory of motion	Zald and Pardo, Mosier and Bereznaya, Suzuki et al.

Initial studies in healthy volunteers described the midline swallowing muscles as bilaterally represented in the motor cortex, but in an asymmetric manner [12]. The Authors highlighted that, in the majority of subjects, pharyngeal and esophageal projections from one hemisphere tended to be larger than those from the other one, suggesting a bilateral but asymmetric motor representation for swallowing musculature between the two hemispheres independently from handedness.

Using a complex paradigm, Hamdy et al. conducted a functional study of the cerebral loci that processes the human volitional swallowing with Positron Emission Tomography (PET) activation imaging, coupled in the same subjects with the single-pulse TMS with EMG recording in order to map the cortical motor representation of pharynx [32]. They firstly confirmed that the normal cortical representation of swallowing is bilateral and asymmetrical; moreover, the findings of sensorimotor cortex lateralization with PET were concordant with the cortical motor representation findings from TMS mapping, suggesting that also the motor aspects of swallowing show some degree of cortical asymmetry.

Lateralized cortical systems has also been indirectly investigated with the dual-task paradigm, by comparing the performances obtained at “baseline” with those obtained while performing specific tasks such as the finger-tapping, the line orientation and the repetition silent [33]. Swallowing was measured at baseline and during each of the three tasks to identify the different contribution of each hemisphere. Swallowing resulted to be impaired by concurrently performing the silent repetition, which mainly activates the left hemisphere, along with the line orientation, a visuospatial process that mostly activates the right hemisphere. Both the finger tapping of the right hand and that of the left hand, that selectively activate their contralateral sensorimotor cortex, interfere with swallowing. Moreover the silent repetition leads to a significant change in the volume of the bolus, while the line orientation seems to have a negative impact on the number of swallows.

Even though it is now widely accepted the existence of a dominant hemisphere for swallowing in each individual, which is however independent from being left or right-handed, it has been not identified a hemisphere uniquely related to dysphagia so far; Notwithstanding, it has been suggested an association between the left hemisphere and the coordination of the oral voluntary phase, and between the right hemisphere and the pharyngeal automatic phase. In this light, evidences from literature suggest that there is an increased tendency for the pharynx to be involved, whether the damage is limited to the right hemisphere [34]; it is also intriguing that some Authors also described an asymmetrical cerebellar activation during swallowing, with a strong lateralization to the left cerebellar hemisphere and/or vermis; this seems to confirm the aforementioned hypothesis that the left brain is mostly involved in the coordination of the oral voluntary phase, while the right hemisphere seems to give a more relevant contribute to the pharyngeal automatic phase.

LESION LOCATION

From a historical point of view, for many years it was thought that a stroke must involve the brainstem or both hemispheres in order to cause dysphagia.

Since many studies over the last three decades have highlighted that also a single unilateral stroke is able to cause a swallowing impairment [34], many efforts have been made to identify those cortical and subcortical region whose damage could affect swallow mechanisms.

The importance of CPG in swallowing control is confirmed by the high incidence of dysphagia in patients with Wallenberg syndrome or lateral bulbar syndrome. Ischemia occurring at this level affects the nucleus of the solitary tract, the nucleus ambiguus and nuclei of the cranial nerves involved in swallowing. The impairment of the nucleus of the solitary tract can affect both the start and the synergy of the pharyngeal phase of swallowing; moreover, dysphagic patients with lesions at this level, have a hypoactivation of those brainstem areas resulting in a swallow impairment which mostly concerns the pharyngeal phase. Otherwise, the damage of the nucleus ambiguous leads to paralysis of the ipsilateral pharyngeal muscles, laryngeal and soft palate.

Flowers et al. [26] highlighted that other brainstem areas, such as the midbrain, the pons, and the lateral and medial portions of the bulb, are strongly correlated with post-stroke dysphagia. They reported as associated with dysphagia midbrain lesions in 6% of cases, pontine lesions in 45%, injuries in the medial bulb in 40%, and in the lateral bulb in 57% of cases; hence there would be a greater risk of swallowing dysfunction after pontine and bulbar stroke.

Also stroke involving insula seems to have a pivotal role in causing dysphagia, since lesions occurring at this level seem to be related to buccofacial apraxia and to a pharyngeal dysphagia strongly associated with aspiration [35].

Daniels and colleagues [27] also claimed that stroke lesions involving the insula may cause dysphagia by means of the disruption of the cortical-subcortical pathway which connect the anterior insula, both the primary and the supplementary motor cortex, the ventroposterior medial nucleus of the thalamus and the nucleus of the solitary tract as well, all structures entrusted in the integrated mechanisms of oropharyngeal swallowing.

It was also observed that basal ganglia, and the associated neural pathways as well, play a role in the pathogenesis of post-stroke dysphagia by means of the progressive involvement of excitatory and inhibitory corticobulbar fibres. In this view, basal ganglia functionally connect the cerebral cortex and thalamus by providing a gate for sensory input entrusted in the motor control of the swallowing. Hence lesions of the basal ganglia and internal capsule are primarily associated with disorders of oral movement control, due to a premature leak of the bolus into the valleculae or the pyriform sinuses [12].

Since the last years, at a subcortical level, it has been a growing attention for the periventricular white matter [36]: the anterolateral portion of periventricular white matter contain both corticospinal descending motor fibers for the mouth and face, and ascending somatosensory ones as well. These ascending and descending fibers are embedded in an intrahemispheric cortico-cortical pathways: they cross the descending corticospinal fibers from the mouth/face representation within the ventrolateral precentral gyrus (motor cortex), and interconnect with specific cortical, subcortical, and brainstem regions involved in swallowing.

To date the role of cerebral small vessel disease (i.e., lacunar infarcts ad white matter hyperintensities) in acute post-stroke dysphagia is still unclear. Notwithstanding it has been suggested that disruption of cortical-cortical and cortical-subcortical white matter connections may increase the risk of dysphagia and aspiration by lowering the threshold of input to the medullary swallowing center.

As far as it concerns cortical lesions, it is reported that a stroke that damages the contralateral precentral gyrus can produce deficits in motor control of the face, lips and tongue and compromise the pharyngeal peristalsis. This type of injury also cause deficits in attention and concentration, hence leading to alterations in the control of the swallowing.

Otherwise, despite several cortical areas have been described as related with swallowing impairment, a definite role in causing dysphagia has been not completely understood for all of them so far. Table 2 shows dysphagia regions of interest as identified by a recent meta-analysis [29]; Authors reported both those areas for which there is a clear implication, identified by most studies, both those ones whose role is still under discussion.

Table 2. Cortical regions involved in swallowing

Region of Interest (ROI) Selected for Analysis and Hypothesized Role in Swallowing		
ROI	Role	References
Primary Somatosensory, Primary and Supplementary Motor cortices (BA 1, 2, 3, 4, and 6)	Cortical processing of swallowing, including motor regulation and execution and sensorimotor control	Hamdy et al., Martin et al., Mosier and Bereznaya
Anterior cingulate (BA 24 and 32)	Higher order motor processing: swallowing movement planning and execution	Hamdy et al.
	Cognitive perceptual processes (attention and response selection)	Martin et al.
Orbitofrontal cortex (BA 10, 11, 12, 44, 45, 47)	Unclear	Mosier et al.
Parieto-Occipital cortex (BA 7,17, 18, 40)	Sensory processing of swallowing	Hadmi et al., Kern et al.
	Task-cue processing (not swallowing per se)	Toogood et al.
	Movement planning and execution	Mosier and Bereznaya
Temporopolar cortex (BA 22 e 38)	Unclear	Mosier et al.
Insular Cortex	Processing of gustatory input	Daniels et al., Daniels and Foundas
	Intraoral sensory modulation	Mosier et al.

In conclusion, these evidences from the literature suggest that swallowing function is provided by a complex neural network bilaterally involving the perirolandic sensorimotor cortex which, together with other cortical structures, projects and receives afferences to the brainstem’ CPG, through the basal ganglia, the thalamus and the periventricular white matter as well.

CPG provides in turn the basic motor plan for pharyngeal swallowing, and coordinative interactions between swallowing and respiration as well [22], by means of its compact clustering of bilateral cranial nerves nuclei and interneuronal network; trough this network, sensory information entering the brainstem from the peripheral structures is integrated into a swallowing motor plan, and then transmitted to the motor nucleus for execution.

Finally, it is worthy of note that, despite the large number of studies, what cortical and subcortical areas are clearly related to dysphagia are not fully clarified so far, nor is it clear how and where their input are integrated and elaborated in order to create a voluntary and functional control of swallowing.

DIAGNOSTIC ISSUES

As mentioned above, there is a great variability among reported post-stroke dysphagia incidence between studies, thereby resulting in a conflicting data concerning dysphagia risk factor, swallowing impairment recovery, and its clinical and neuroanatomical correlate as well.

This wide range of variability is mainly due to both diagnostic criteria and assessment methods issues.

With regard to the criteria used for post-stroke dysphagia diagnosis to be made, it is worth considering what exactly is meant by the term dysphagia: oropharyngeal dysphagia is probably best defined as a disruption of bolus flow through the mouth and pharynx, as resulting from multiple measures of bolus flow, structural movement and patient perception as well [37].

Several studies aimed to investigate post-stroke dysphagia actually referred to aspiration, a related but distinct term which means the penetration of food material into the airways and below the true vocal cords.

From this point of view this seems somehow misleading, firstly because it is well known that dysphagia may occurs without aspiration; moreover, even with an instrumental evaluation, the incidence of dysphagia may be overestimated if its identification is based on laryngeal penetration or single occurrences of aspiration [28], as also healthy subjects may exhibit laryngeal penetration and sometimes aspiration as well [20].

Finally, by focusing on the overt sign of aspiration to diagnose post-stroke dysphagia, such as cough or voice change, the aforementioned silent aspiration would be missed, with a relevant increase in relative risk of pneumonia and a poorer clinical outcomes.

Concerning the implied diagnostic methods, the post-stroke dysphagia incidence is reported to be lower with the adoption of less accurate and precise screening tests (37%-45%), higher when clinical tests are used (water swallow test, 51%-55%) and even higher when dysphagia diagnosis is based on instrumental tests (fiberoptic endoscopic evaluation of swallowing or videofluoroscopy, 64%-78%) [3].

Among the instrumental assessment tools, videofluoroscopic evaluation (VSE) and fiberoptic endoscopic evaluation of swallowing (FEES) are thought to be the current gold-standard. These techniques are equally sensitive to the detection of post-stroke aspiration and pneumonia and likewise useful in the evaluation of oropharyngeal dysphagia [38].

However, these tests have several practical limits. VFS is a relatively complex task both to perform and to interpret; test-test variability has been found within normal subjects undergoing this exam and interjudge agreement between different radiologists for VFS assessment can be very variable, probably reflecting their different skill or experience [39, 40]. Moreover for VFS to be undertaken, patients have to cooperate or at least be able to sit upright, which does not replicate physiological conditions. Further limitations are imposed by radiation exposure involved which, although regarded as acceptable, makes it an inappropriate test to repeat frequently to monitor changes [38].

Otherwise, FEES is a safe and well tolerated assessment conducted at bedside thereby allowing timely scheduling for both the endoscopist and patient [41, 42]; it is however an operator-dependent examination requiring both operator and specialized equipment expertise; besides it provides limited information about the oral and esophageal stages since aspiration cannot be seen directly, being inferred from residue left after swallowing or ejected after coughing [43].

Given the aforementioned limitations of those instrumental tools and considering that both VFS and FEES are not available in many clinical settings in which stroke patients are managed, there is a need of a valid, reliable, and feasible screening tool for the early assessment of dysphagia.

In this point of view, bedside clinical assessment of swallowing (BSA) has become a widely used initial test to identify those patients who would benefit from a more detailed assessment of their swallowing, such as VFSS or FEES [44].

By definition, this should be simple, quick to perform at bedside and sensitive enough to accurately identify stroke patients with risk of swallowing impairment. As a matter of fact a high sensitivity is required because of the increased morbidity and mortality associated with aspiration, which necessitates requirements for low false-negative results [1].

Nonetheless, the Joint Commission recently excluded swallowing screening as a performance measure for Primary Stroke Center certification [45]; moreover, although there are currently many bedside tests, clinical bedside assessment has been shown to miss up to 40% of those patients who aspirate; this is mostly because of the lack of systematically defined standards for what constitutes a valid swallowing screening tool, being still unclear which of these have the best psychometric and feasible properties and are easy to administer.

The following are the features most widely used in the literature as a part of the bedside evaluation of dysphagia.

Water Swallow Test (WST)

This test used water administered in various aliquots and ways as a trial swallow, while monitoring coughing and choking during and after swallowing. Indeed in the literature, the number of trials varied, together with water volumes administered which ranged from 5 to 150 mL. Different features has been also monitorized during WST, depending on the different study designs; Leder and colleagues found that the presence of 2 of 6 features (dysphonia, dysarthria, abnormal volitional cough, cough after swallow, abnormal gag reflex, and voice change after swallow) predicted greater dysphagia severity on VF [42]. When analyzing studies that used WST to evaluate aspiration and/or penetration [39, 44, 46-48], sensitivity ranged from 27% to 85%, while specificity ranged from 50% to 88% [49]. Several Authors [50-52] also used different viscosities textures (i.e., liquids, semi-solids and solids) while performing WST; although they reached an higher sensitivity and specificity, the assessment feasibility became lower because of the need of both considerable quantities of text materials and the time required for the test to be done.

Bulbar Function Assessment

WST effectiveness can otherwise be increased by careful assessment of bulbar function (i.e., abnormalities of pharyngeal sensation and reduced cough reflex. This is mostly because cough and wet voice in response to a WST, together with abnormal palatal sensation, are thought to be essential predictors of prolonged dysphagia [53], aspiration [1] and particularly useful in detecting silent aspiration [54].

Smithard and colleagues also identified weak voluntary cough after drinking different volumes of water, along with impaired consciousness level, as independent predictor for aspiration [39].

Moreover in acute stroke patients with dysphagia, a sensory deficit in the pharynx and supraglottic has been demonstrated by now. This sensory deficit is thought to play a pivotal role in the development of aspiration both directly, by reduced stimulus detection and discriminative capacity in the laryngopharynx, and indirectly, in impaired triggering of motor actions [53].

In this light many Authors searched for a reliable clinical or instrumental tool, which could allow a valid bulbar function assessment. Smith-Hammond and colleagues found that in stroke patients, objective measures of voluntary cough (i.e., duration, volume and peak flow of the inspiration phase, compression phase duration, peak flow and rise time for the expulsive phase, and volume acceleration) are strongly related to aspiration [55]; in this study it was also stated that subjective assessments of voluntary cough had otherwise poor performance.

Sensory capacity of the laryngopharynx has been also determined by endoscopically delivering discrete air pulse stimuli directed at mucosa innervated by the internal branch of the superior laryngeal nerve. Given that air pulses test has been proven to detect laryngo-pharyngeal sensory deficits also in stroke patients with no clinical dysphagia [41], this examination might be essential for the detection of the so-called silent aspirators. Notwithstanding, it also appears to be not such a practical and reliable tool mostly because of it requires both an endoscopically delivered stimulus and an air pulse instrument.

Pulse Oxymetry and Silent Aspiration

Although pulse oxymetry has been criticized in the literature as a valid screening tool [56, 57], many studies focused on oxygen desaturation to evaluate aspiration/penetration since it has been shown to detect silent aspiration [44, 46, 52]. Depending on the decrease between the baseline oxygen saturation and the minimum saturation during the 120 sec, a pulse oxymetry decrease of greater than or equal to 2% has been widely considered as the endpoint. The sensitivity of this test so ranges from 56% to 87% while specificity from 39% to 97%, both depending on the different studies [39, 48]. Moreover, two different studies tested a combination of a swallow and oxygen desaturation test, by using coughing, choking, voice change or ≥2% desaturation as the endpoints [44, 46].

With this paradigm, they reached a sensitivity of 94% and 98%, and a specificity of 63% and 70%, respectively [58]. In agreement with these data, in a recent review performed by Bours and colleagues it is stated that, by considering both the best psychometric properties and the feasibility of the screening methods, WST combined with pulse oxymetry (considering choking, coughing, voice change and desaturation ≥2% as fixed endpoints), should be considered, to date, the most promising results as a screening tool in terms of validity, reliability and feasibility [49].

Nevertheless, although the most important international guidelines stress the importance of Screening of swallowing before the administration of food, liquid, or medication in patients with stroke, they do not recommend a specific screening method so far [59].

There is not even consensus in the literature, regarding either the best type of swallowing assessment to use, i.e., clinical or instrumental, or what specific characteristics acute stroke patients should have in order to benefit from either type of testing [42].

Hence WST appears as a simple tool, quick to perform at bedside and sensitive enough to identify stroke patients with risk of swallowing impairment.

CLINICAL AND NEUROANATOMICAL PREDICTORS OF POST-STROKE DYSPHAGIA

A recent study was conducted in order to assess clinical and neuroanatomical predictors of dysphagia following stroke [60]: the main purpose of the study was to investigate a sample of consecutive patients with ischemic and hemorrhagic stroke to evaluate incidence, prognosis, clinical and neuroradiological correlates of post-stroke dysphagia. Consecutive patients with acute ischemic or hemorrhagic stroke were consecutive recruited and followed-up after 14 days, in order to assess post-stroke swallowing impairment; according to the swallowing function, patients were then classified as: a) persistent dysphagic b) non-persistent dysphagic or c) non-dysphagic. The role of leukoaraiosis in the pathogenesis, persistence and prognosis of post-stroke dysphagia is also discussed.

Patients who presented dysphagia were significantly older than those without; thus, elderly patients with stroke may have more swallowing alterations due to the reduced cough reflex and alteration of swallowing/ breathing coordination [61].

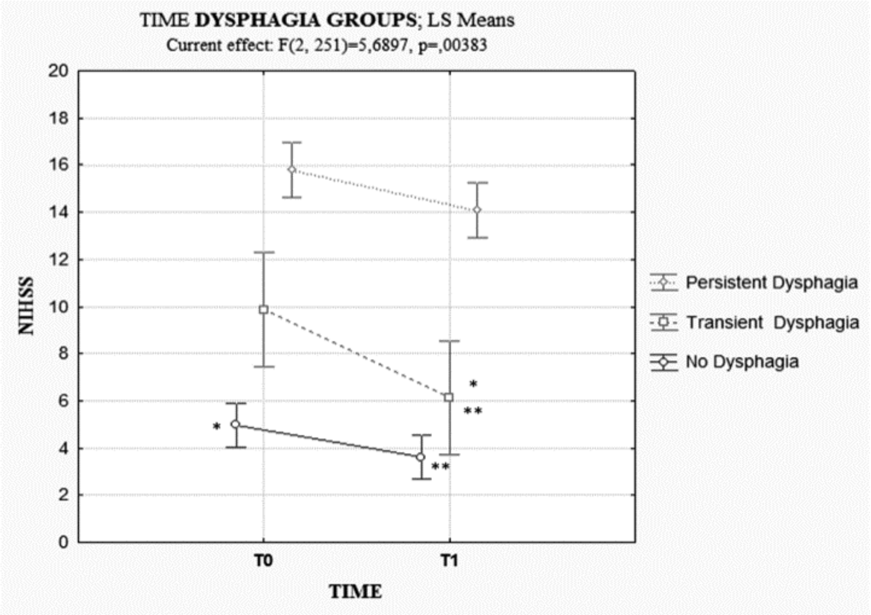
Dysphagia was influenced by the lesion size, but no differences were identified in the involvement of right or left hemisphere. In this view, the majority of studies performed on stroke patients did not observe any relation between the presence of dysphagia and side of hemispheric location [62-64]. However, it is known that lesions located in the right hemisphere may cause more pharyngeal changes and that lesions in the left hemisphere cause more changes in the oral phase of swallowing [65].

The NIHSS score at admission was higher in dysphagic patients. With this regard, the three different pattern of dysphagia (i.e., absent, transient or persistent) were compared over time regarding the stroke severity at admission, data from the ANOVARM after Tukey HSD post hoc analysis showed (Figure 1):

1. Patients with persistent dysphagia (PD) vs. patients with transient dysphagia (TD):
    - PD showed a significantly higher T0 and T1 NIHSS score with respect to both T0 ($p < 0.001$, $p = 0.023$ respectively) and to T1 ($p < 0.001$, $p < 0.001$ respectively) NIHSS scores of TD.
1. Patients with persistent dysphagia (PD) vs. no dysphagic patients (ND):
    - PD showed a significantly higher T0 and T1 NIHSS score with respect to both T0 ($p < 0.001$, $p < 0.001$ respectively) and to T1 ($p < 0.001$, $p < 0.001$ respectively) NIHSS scores of ND.
2. Patients with transient dysphagia (TD) vs. no dysphagic patients (ND):
    - TD showed a significantly higher T0 NIHSS score with respect to both T0 ($p = 0.002$) and to T1 ($p < 0.001$) NIHSS scores of ND, whereas T1 NIHSS of TD did not differ from both T0 ($p = 0.95$) and to T1 ($p = 0.38$) NIHSS scores of ND.

In other words, stroke severity was significantly higher in patients with persistent dysphagia with respect to those with transient dysphagia or without dysphagia, both at admission and at T1. Moreover, those patients with a transient pattern of dysphagia showed a sensibly improved NIHSS score at T1, which became similar, on average, to that of patients without swallowing impairment upon admission.

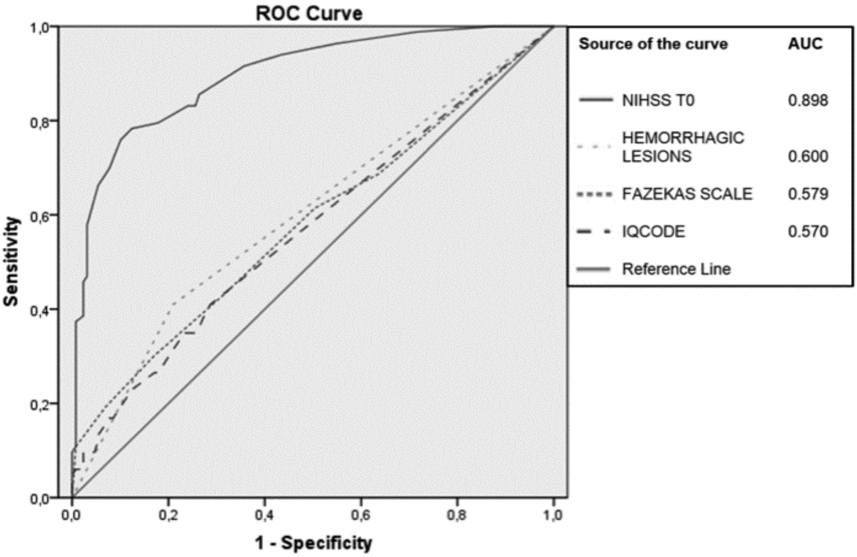
In the multivariable logistic analysis, stroke severity at admission was found to be an independent predictor of a persistent pattern of post-stroke dysphagia. The multivariable logistic model also allowed to represent ROC (receiver operating characteristics) curves and evaluate the predictivity of each significant variable using the AUC (Area Under the Curve) value. In this view, looking at ROC analysis, the T0 NIHSS score of 11.5 was found to be the best predictive value of persistent dysphagia, with a specificity of 90.1% and a sensitivity of 72.4%; the reported AUC was 0.898, which means that stroke severity at admission allowed to correctly classifying about 90 of 100 dysphagic patients in a persistent or non-persistent pattern. Hence, a NIHSS ≥ 12 would be suggested as cut-off value in order to predict, upon admission, those patients who will probably remain dysphagic after 14 days follow-up. Figure 2 shows the ROC curves and the respective AUC.



Tukey post hoc analysis showed a significant difference between all groups (longitudinally compared among NIHSS) but: *p = 0.95, **p = 0.38.
See the text for the details.

Figure 1. ANOVA for Repeated Measures.

Dysphagia was also more frequent in patients with hemorrhagic stroke; there are little available data in the literature reporting a different risk factor for dysphagia depending on stroke type. Paciaroni et al. [63] found, in a univariate comparison, an increased frequency of dysphagia in hemorrhagic stroke patients compared to ischemic ones. They suppose that it could be related to stroke severity. Our results confirm this datum, since the Spearman's Rank Correlation revealed a strong statistical dependence between stroke severity at admission and hemorrhagic stroke. Notwithstanding, looking at logistic regression analysis, the stroke type also appears an independent predictor for post-stroke dysphagia (OR 2.5); nonetheless hemorrhagic patients present a more than three-folded risk of persistent dysphagia. Hence, further research is needed in order to investigate a possible intrinsic mechanism linking dysphagia and hemorrhagic stroke.



AUC = Area Under the Curve.

Figure 2. ROC Analysis: persistent dysphagia.

Patients with high IQCODE score, which means a pre-stroke cognitive impairment, had more chances to present swallowing dysfunction; moreover, logistic regression analysis showed that the IQCODE score is an independent risk factor for post-stroke dysphagia and persistent dysphagia as well. These results are in agreement with previous reports which stated that brain lesions that cause decline in cognitive function, such as concentration and attention, may impair the control of swallowing [66].

Dysphagia was more frequent in patients with insular stroke, as well as in those with stroke involving frontal and temporal lobes, and basal ganglia. As a matter of fact, all the aforementioned structures are involved in the neural control of swallowing; in particular, insula is known to be closely connected both with cortical and subcortical pathways involved in the neural control of swallowing. Nevertheless, none of those structures was found to be an independent risk factor for dysphagia. In the case of insular stroke, this seems mostly due to the small number of pure insular stroke.

Otherwise, concerning the others aforementioned structures who failed to reach statistical significance once logistic regression analysis has been performed, this datum could suggest that cortical-subcortical pathways involved in the swallowing function are probably more important in causing dysphagia after stroke than a single structure “*per se*.”

This hypothesis could be corroborated by analyzing the contributions of white matter to swallowing behavior. Interestingly, the Authors observed an independent correlation between post stroke dysphagia and leukoaraiosis severity as well as a strong association between leukoaraiosis and previous cognitive impairment. Recent studies reported that subcortical white matter connections play an important role in the pathogenesis of dysphagia: disruption of cortical-cortical and cortical-subcortical white matter connections seems to increase the risk of dysphagia and aspiration pneumonia by lowering the threshold of input to the medullary swallowing center.

These data, as well as those showing the role of a decline in cognitive function, suggest that swallowing impairment after stroke might be not just a simple pure motor disturbance, but also a more complex phenomenon. In this context, one might speculate that dysphagia may result from the combined effects of an acute focal damage and a preexisting impairment of the subcortical white matter connecting pathways. In other words, we might think of dysphagia as a kind of deficit in executive functions, which we know to be particularly associated with leukoaraiosis and cognitive disorders [67], and of course age [61].

Finally, pneumonia is among the most common medical complication after stroke, affecting up to one third of patients and accounting for nearly 35% of post-stroke deaths [2, 68]. Data from the aforementioned study [60] also suggest that stroke and pneumonia are associated with two-fold: on the one hand, pneumonia appears to be strongly linked to the presence of dysphagia at admission. In fact, among those patients who developed pneumonia during the entire hospitalization, 38 (93%) were dysphagic at admission. On the other hand, the persistence of dysphagia after 14 days was closely linked with the occurrence of pneumonia ($p < 0.001$), with 36 patients (88%) with pneumonia who showed a persistent pattern of dysphagia.

RECOVERY FROM POST-STROKE DYSPHAGIA AND PERSISTENT DYSPHAGIA

The nervous system affected by a stroke damage is thought to reserve a variable ability to somehow reorganize damaged pathways and structures, allowing an effective recovery from swallowing impairment.

In this view, recent findings seem to suggest that the reorganization of cortical maps actually represents a central features in recovery from dysphagia after stroke.

In a MEG study, Teismann and colleagues [69] analysed the swallowing-related activation areas both in stroke patients with dysphagia, in those patients without dysphagia and in healthy control as well. In the acute phase of stroke, it was described:

Patients with hemispheric stroke: while those patients without dysphagia reported an activation of the bilateral hemispheres, patients with post-stroke dysphagia are described to have a decreased cortical activation of the ipsilesional sensorimotor areas, and a near extinguished activation of the contralesional hemisphere; the whole sample of hemispheric stroke patients also showed an activation of the prefrontal cortex (BA 9) which was neither seen in healthy subjects nor in patients with brainstem stroke.

Patients with brainstem stroke: in both patients with and without dysphagia it was observed a reduced cortical activation, due to cortical compensation of a subcortical caused dysphagia, as well as a strong right hemispheric lateralization; the Authors speculated that this phenomenon may be related to “diaschisis.” This concept was developed by Von Monakow in 1914 and refers to the fact that the acute lesion may cause a damage of other areas functionally linked.

In healthy controls they observed a bilateral activation, mostly involving both the primary and supplementary motor cortex (BA 4 and 6). Other activation areas was represented by the primary and secondary sensory areas (BA 1, 2, 3, 4, 5, 6 and 7).

Hence, in conclusion, a strongly reduced cortical activation was bilaterally observed in stroke patients with dysphagia, probably due to an initial cortical shock after stroke.

What is absolutely remarkable, is that the activation of contralesional areas changed over time: in contrast to a strongly reduced activation in the acute phase, in the subacute phase of stroke (mean 12 days after stroke) this pattern changed to an increased brain activation of the contralesional hemisphere [69].

This datum seems to be crucial in order to understand the complex mechanisms leading to recovery from swallowing impairment after stroke.

In this point of view, it has been postulated that the recovery mechanisms from post-stroke dysphagia involve changes in neural networks already different from those described in the motor recovery after stroke.

After motor stroke, a reduced cortico-cortical connectivity in the affected hemisphere has been already described, as well as disinhibition phenomena (i.e., increased task-related BOLD activity) in the unaffected (i.e., controlesional) hemisphere [70]. Notwithstanding, the role of the controlesional primary motor cortex (M1) for motor recovery remain somehow controversial: it is reported that enhanced activity in controlesional M1 might even exert a negative influence on the motor recovery of the paretic hand, probably due to transcallosal inhibition mechanisms between the primary motor cortex of both hemispheres. This "inter-hemispheric inhibition" is otherwise confirmed by several TMS-fMRI studies which show that stroke patients with motor impairment may benefit from controlesional M1 inhibition [71]. Hence, it is suggested that a strong engagement of the unaffected hemisphere may represent an indicator of inefficient cortical reorganization, thereby exerting a detrimental effect on motor recovery after stroke.

On the contrary, recent data shows that recovery from post-stroke dysphagia follows a reorganization of the unaffected motor cortex.

Hamdy and colleagues [72], by coupling fMRI analysis with TMS-evoked EMG responses, described a different compensatory reorganization between hemispheres during the recovery of swallowing and limb function: while improvements in limb function were associated with an increase in cortical representation in the damaged hemisphere, patients who recovered from dysphagia, showed an increased pharyngeal representation in their cortical maps over the unaffected hemisphere, both at one and at three month after stroke, without change in the affected hemisphere [37,62]. In contrast, those patients who remained dysphagic, did not show any variation in their pharyngeal map.

The most interesting findings is that this cortical change seems to appear before any improvement in swallowing function, suggesting that the change in cortical excitability for pharynx is not merely the consequence of the normal swallow activity resumption, but it actually represents the drive for dysphagia recovery.

Moreover, they also found that pharyngeal responses from the unaffected hemisphere showed a marked increase in both the amplitude and area of representation over time, without concurrent shift in latency; this datum suggest an alteration in the excitability of cortex rather than more caudal brain regions, such as brainstem central pattern generator or bulbar motoneurons.

It is intriguing that it was also described a different compensatory reorganization between hemispheres during the recovery of swallowing and limb function.

It could be then speculated that this finding supports the hypothesis of a bilateral and asymmetric representation of swallowing: since swallowing musculature, unlike limb musculature, is represented on both hemispheres, although a damage to the dominant hemisphere for swallowing may cause dysphagia, there remains within the intact hemisphere the capacity for reorganization, by exerting a sufficient control over brainstem centers to drive swallowing recovery.

Notwithstanding, about half of stroke patients remain dysphagic after seven days from symptoms onset, and approximately 13-15% [73] of patients has persistent dysphagia after six months.

To date, it is unclear why some stroke patients develop a persistent swallowing impairment after stroke or, in other words, if the failure in recovery from dysphagia is due to a stroke involving the dominant swallowing hemisphere, rather than a swallowing key region, or even to a less efficient contralesional hemisphere activation, perhaps due to an insufficient bilateralism in cortical representation of swallowing.

In this view, many efforts have been made in order to identify those patients who are at high risk for developing a persistent dysphagia after stroke, and therefore to identify those patients who may benefit from a more detailed assessment of their swallowing function.

Nevertheless, clinical and neuroradiological correlates of post-stroke dysphagia remain still unclear, and even fewer data are available on predictors of persistent dysphagia so far.

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Chapter 3

DYSPHAGIA AND PNEUMONIA IN ACUTE INTRACEREBRAL HEMORRHAGE

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ABSTRACT

Dysphagia, defined as difficulty in the passage of solids or liquids from the mouth to the stomach, is common after stroke. Aspiration is a major risk factor for the development of pneumonia. Dysphagia and pneumonia in intracerebral hemorrhage (ICH), however, have not been well investigated. Incidence, mechanism, and treatment of dysphagia and pneumonia in acute ICH are discussed.

INTRODUCTION

A variety of medical complications, such as epileptic seizure, dysphagia, infection, falls, pressure sore, thromboembolism, pain, and depression, are frequent in acute and subacute stroke, and are associated with longer hospital stay, higher medical expense, and poor outcome. Among different types of post-stroke complications, dysphagia and its sequelae, including malnutrition, dehydration, suffocation, and aspiration pneumonia, are directly related to mortality, and therefore, of major concern. In this chapter, incidence, risk factors, mechanism, and treatment of dysphagia and pneumonia after intracerebral hemorrhage (ICH) are described.

DYSPHAGIA AFTER ICH

Oropharyngeal dysphagia or swallowing disorder is common after stroke. Post-stroke dysphagia is frequent soon after the onset of stroke, and naturally improves (Smithard et al. 1997). The presence of dysphagia can be identified on the basis of clinical or instrumental examinations, such as the water swallowing test, videofluoroscopy, and fiberoptic endoscopy. Incidence rates are reported to be between 29% and 67% in acute stroke patients (Gordon et al. 1987, Smithard et al. 1997, Mann et al. 1999, Martino et al. 2005). Some of the variability is related to differences in the timing and method of swallowing assessment. Studies using water swallowing tests to detect dysphagia tend to yield slightly lower dysphagia rates than studies that used videofluoroscopy (Takizawa et al. 2016). Post-stroke dysphagia is associated with poor outcome and mortality (Smithard et al. 1996).

Oropharyngeal dysphagia in ICH patients is still largely uncertain. Existing studies have simply investigated patients with ischemic stroke or excluded severely affected patients. Because they used clinical or radiological swallowing evaluations in diagnosing oropharyngeal dysphagia (Smithard et al. 1997, Mann et al. 1999), the subjects needed to be stable and conscious to cooperate. Many ICH patients were thus excluded from many studies that investigated dysphagia following stroke.

Paciaroni et al. (2004) examined 406 patients with acute stroke who were conscious (Glasgow Coma Scale ≥10) and medically stable and had no history of previous swallowing impairment or a medical condition that could affect swallowing function. In their study of dysphagia following stroke, 31 of 63 (42.9%) ICH patients were assessed as dysphagic. This incidence was significantly higher than in patients with ischemic stroke (32.1%, 110/343, $p = 0.01$).

Maeshima et al. (2014) restricted inclusion to patients with thalamic hemorrhage and reported that bedside swallowing assessment using modified water swallowing test was abnormal in 55% (62/113) of the subjects. Their study has the largest acute ICH population at present.

Malnutrition and Dehydration

Malnutrition and dehydration are common after stroke due to swallowing impairment, immobility, and communication difficulties (Bhalla et al. 2000, Finestone et al. 2001, Kelly et al. 2004). Malnutrition and dehydration increase during the first week of hospitalization and are associated with poor outcomes including increased complications and mortality (Foley et al. 2006). The nutritional needs of those with hemorrhagic stroke may be higher than previously calculated, and these patients may be at particular risk of malnutrition (Esper et al. 2006).

Risk Factors for Severe Dysphagia after ICH

Percutaneous endoscopic gastrostomy (PEG) is considered in patients with severe dysphagia who cannot effectively tolerate oral intake. Kiphuth et al. (2011) retrospectively studied risk factors for PEG in 208 patients with spontaneous ICH. PEG tube placement was conducted in ventilated patients within 30 days and in spontaneously breathing patients if swallowing did not improve within 14 days. Fifty-one (24.5%) patients received PEG tubes. They found that worse Glasgow Coma Scale measures at the time of admission, occlusive hydrocephalus, mechanical ventilation, and systemic sepsis were independent risk factors for PEG tube placement.

Another retrospective survey on 189 spontaneous ICH patients was conducted by Faigle et al. (2015). In their study, PEG placement was considered when patients had failed bedside and videofluoroscopic swallowing assessment and had failed serial attempts to demonstrate adequate swallowing function. They found that 58 (30.7%) of the studied population received PEG placement and that age, black race, and ICH volume were independent predictors of PEG placement.

In contrast to the above two studies, studies investigating predictors of PEG placement in ischemic stroke cited bulbar palsy or bilateral hemispheric lesions, which could cause pseudobulbar palsy, as risk factors for PEG (Alshekhlee et al. 2010, Kumar et al. 2012), whereas hemorrhage in the lateral medulla oblongata is extremely rare in ICH. Cause of severe dysphagia may slightly differ between ischemic stroke and hemorrhagic stroke.

ALTERNATIVE NUTRITION FOR TREATMENT OF SEVERE DYSPHAGIA

Non-oral or tube feeding (nasogastric tube and PEG tube placement) has become a well-established clinical practice in stroke patients who are dysphagic and at high risk for aspiration or who cannot meet their nutritional needs orally. Stroke is indeed the primary cause of PEG tube placement (Grant et al. 1998, Suzuki et al. 2010), but there is no clear evidence to suggest one route is more beneficial than any other in stroke patients. Moreover, high incidences of chest infection and death are each associated with both methods of tube feeding (Grant et al. 1998, Dennis et al. 2005, Ickenstein et al. 2005). Grant et al. (1998) carried out a retrospective population-based descriptive study employing 81,105 Medicare beneficiaries (age >65) who received PEG tube placement and found overall mortality rate at 30 days was 23.9%, reaching 63.0% at 1 year and 81.3% by 3 years. Suzuki et al. (2010) found median survival of PEG patients was 753 days, and the most frequent cause of death was pneumonia (59%).

Dysphagia Therapy

Several studies have examined the effect of formal dysphagia therapy in stroke patients. Dysphagia therapy usually involves a combination of approaches, including exercises aimed at strengthening muscles and improving movement and coordination. Other strategies include postural changes (reclining sitting posture and chin down position) and multiple swallows. These therapies are usually provided in addition to dietary modifications.

Carnaby et al. (2006) examined the effectiveness of early behavioral interventions in the recovery of swallowing in acute stroke patients. They randomized 306 patients with clinical dysphagia, including 29 ICH patients, to 1 of 3 treatment groups: (1) usual care by attending physician, mainly consisting of feeding supervision; (2) low-intensity swallowing therapy 3 times per week for 1 month with environmental modifications, advice, and dietary modifications; and (3) high-intensity treatment every day for 1 month with effortful swallowing using the supraglottic technique. Of those assigned to usual care, 55.9% returned to normal diet at 6 months as compared with 66.7% in the intervention group (p = 0.07). Behavioral intervention was associated with a reduction in swallowing-related medical complications.

Chest infection due to aspiration was 47.1% for the control and 26.5% for the intervention group ($p < 0.01$). Patients allocated to the intervention group compared with usual care were less likely to be deceased or institutionalized at 6 months.

Elmståhl et al. (1999) examined the effect of dysphagia treatment on nutritional status in stroke patients with dysphagia. Thirty-eight patients received swallowing therapy, including oral motor exercise; swallowing strategies (supraglottic swallowing, effortful swallowing, Mendelsohn maneuver, and thermal stimulation); head and neck positioning; and diet modification. Swallow function as observed on videofluoroscopic swallowing examination improved in 61% (23/38) of the subjects ($p<0.01$). Patients (n=23) who improved their swallowing function increased their albumin levels after treatment from 33.7 g/l ±4.6 to 36.2 g/l ±3.2 ($p<0.01$) and also increased their total iron-binding capacity from 44.4 µmol/l ±13.4 to 52.0 µmol/l ±8.9 ($p<0.01$). Patients (n=15) who did not respond to therapy had decreased body weight from 75.5 kg ±13.3 to 72.2 kg ±11.8 ($p<0.05$).

There is one study concerning dysphagia treatment in ICH patients at present. Takahata et al. (2011) retrospectively investigated the effect of intensive oral care and early initiation of oral feeding in 219 acute ICH patients. The first group (n=90) was admitted before implementation of an early intervention program delivered mainly by nurses. The second group (n=129) was admitted after implementation. The intervention program included screening prior to initiation of oral intake using pudding or jelly (not water), intensive oral care, postural adjustment during feeding with chin tuck, and advancement of the diet, as appropriate. The proportion of patients who could tolerate oral feeding was significantly higher in the early intervention group compared with the control group: 112 (86.8%) vs. 61 (67.8%), $p<0.001$. After adjusting for baseline imbalances, early intervention was still associated with oral feeding tolerance (odds ratio [OR] 4.42, 95% confidence interval [CI] 1.81-10.80; $p=0.001$). Incidence of chest infection was lower in the early intervention group compared with the control group: 27/129 (20.9%) vs. 32/90 (35.6%); OR 0.48, 95% CI 0.26-0.88; $p=0.016$.

Pneumonia after ICH

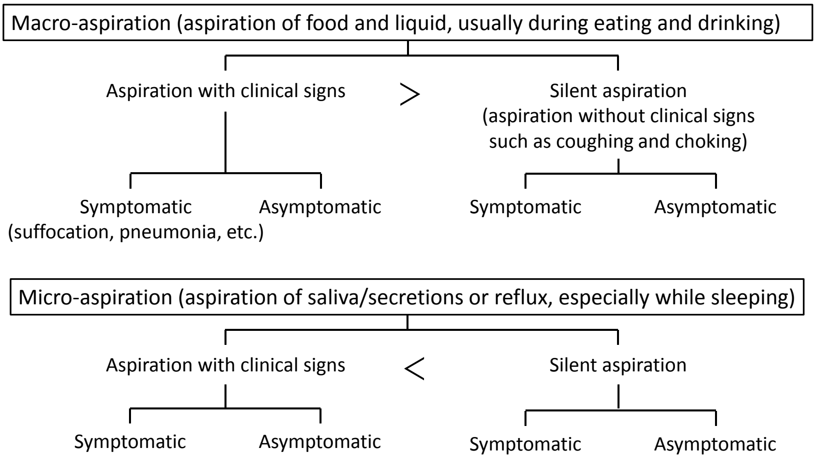
Pneumonia is one of the most frequent medical complications of stroke and is associated with poor outcome as with dysphagia, leading to a threefold increased risk of death (Langhorne et al. 2000, Katzan et al. 2003, Kishore et al. 2015, Smith et al. 2015). The reported incidence of respiratory infection is nearly 15% to 20%, ranging from 5% to 50% depending on the definition of pneumonia, diagnostic method, setting, and timing of diagnosis. There have been no agreed-upon gold standard or reference standards for diagnosis of pneumonia in stroke unit care, and various terminologies are used to describe the spectrum of lower respiratory tract infections complicating stroke (Kishore et al. 2015). Chest infection according to Mann criteria (Mann et al. 1999) has been one of the widely used terms for describing infectious complication in the respiratory system after stroke. Diagnosis of chest infection is based on the presence of ≥3 of the following variables: fever (>38° C); productive cough with purulent sputum; abnormal respiratory examination (tachypnea [>22/min], tachycardia, inspiratory crackles, bronchial breathing); abnormal chest radiograph; arterial hypoxemia (PO_2 <70 mm Hg); and isolation of a relevant pathogen (positive gram stain and culture). This definition, however, may include upper respiratory tract infection such as tracheobronchitis other than pneumonia, because abnormal chest radiograph is not necessarily required.

Recent recommendations for diagnosis of stroke-associated pneumonia (SAP) are as follows: (1) SAP is the recommended terminology for the spectrum of lower respiratory tract infections within the first 7 days after stroke onset; (2) modified CDC criteria are proposed for SAP as follows—probable SAP: CDC criteria met, but typical chest x-ray changes absent even after repeat or serial chest x-ray; definite SAP: CDC criteria met, including typical chest x-ray changes; (3) there is limited evidence for a diagnostic role of white blood cell count or C-reactive protein in SAP; and (4) there is insufficient evidence for use of other biomarkers (e.g., procalcitonin) (Smith et al. 2015).

Post-stroke pneumonia or SAP usually occurs early after stroke onset and is more prevalent in tube-fed patients who do not consume food or liquid orally than in those with oral feeding (Dziewas et al. 2004, Langdon et al. 2009, Teramoto 2009). Mechanical ventilation, tube feeding, aspiration, and tracheostomy are generally thought to be associated with increased risk of the development of pneumonia not simply in ischemic stroke but in ICH (Alsumrain et al. 2013, Divani et al. 2015). Although most SAPs have been believed to result from aspiration (Johnston et al. 1998), tube-fed patients or mechanically ventilated patients are unlikely to aspirate anything other than saliva/secretions or reflux, yet experience significantly higher rates of pneumonia than those fed orally. Therefore, it should be natural to consider that micro-aspiration of colonized saliva/secretions, which occurs in dysphagic patients when they are sleeping or under mechanical ventilation, is more relevant in the development of SAPs than macro-aspiration, which takes place while taking noncolonized food or liquid orally. (See the figure for the explanation of macro-aspiration and micro-aspiration.)

Recently it has been realized that CNS injury, including stroke, induces immunodepression, and that this is the mechanism by which CNS injury leads to secondary immunodeficiency (CNS injury-induced immunodepression) and infection (Meisel et al. 2005). Laboratory and clinical data have shown stroke-induced immunodepression plays an important role in the development of infectious complications in ischemic stroke (Dirnagl et al. 2007, Hannawi et al. 2013), whereas immunodeficiency in hemorrhagic stroke has not been well elucidated. The impact of stroke-induced immunodepression in ICH has to be further studied.

Another pulmonary complication associated with aspiration is aspiration pneumonitis (Marik et al. 2001, Kumar et al. 2010). Aspiration pneumonia is caused by inhalation of colonized bacteria that seed pharyngeal material or that reside in the gingival crevices, whereas aspiration pneumonitis, a sterile lung injury, is caused by aspirating acidic gastric contents, typically when vomiting and sleeping. Bacteriological finding of aspiration pneumonitis is initially sterile, with possible subsequent bacterial infection. Aspiration pneumonitis is, therefore, often self limited and does not require antimicrobial therapy; occasionally it causes severe lung inflammation and acute respiratory distress. Aspiration pneumonia is literally a bacterial infection and requires empirical antimicrobial treatment for gram-negative bacilli and gram-positive cocci.


Macro-aspiration (aspiration of food and liquid, usually during eating and drinking)
Aspiration with clinical signs
>
Silent aspiration
(aspiration without clinical signs
such as coughing and choking)
Symptomatic
(suffocation, pneumonia, etc.)
Asymptomatic
Symptomatic
Asymptomatic
Micro-aspiration (aspiration of saliva/secretions or reflux, especially while sleeping)
Aspiration with clinical signs
<
Silent aspiration
Symptomatic
Asymptomatic
Symptomatic
Asymptomatic

Macro-aspiration and micro-aspiration.

Prevention of Pneumonia after ICH

A formal screening procedure for dysphagia is strongly recommended before initiation of oral intake to reduce the risk of pneumonia (Hemphill III et al. 2015). In a prospective multicenter observation study, use of a formal screening protocol for dysphagia (e.g., water swallow test) for all patients admitted with ischemic stroke was associated with a significantly reduced risk of pneumonia compared with no formal screen. The pneumonia rate of sites with a formal dysphagia screen was 2.4% versus 5.4% of those without a screen, a 3% absolute risk reduction (Hinchey et al. 2005). Another study including both ischemic and hemorrhagic stroke also showed that higher adherence to the dysphagia screen was associated with significantly decreased incidence of hospital-acquired pneumonia (OR 0.43, 95% CI 0.255-0.711; p = 0.0011) (Titsworth et al. 2013). Various measures such as nothing by mouth, diet modification, and postural change are given in those who failed the screen to reduce aspiration. However, there has been no clear explanation of why higher adherence to the formal swallow screen reduces risk of pneumonia when patients with non-oral feeding are especially susceptible to pneumonia.

Oral hygiene care (OHC), using either a mouth rinse, gel, toothbrush, or combination, is an established practice to reduce risk of pneumonia in the elderly (Yoneyama et al. 1999, Teramoto 2009, Shi et al. 2013). The importance of OHC has been recognized in ICUs, and OHC could lead to reduced incidence of ventilator-associated pneumonia and hospital-acquired pneumonia (Fields 2008, Yao, et al. 2011, Klompas et al. 2014, El-Rabbany et al. 2015). Wagner et al. (2016) compared the proportion of pneumonia cases before and after implementation of a systematic OHC intervention in patients hospitalized with acute ischemic stroke or intracerebral hemorrhage in a large, urban academic medical center and found that systematic OHC use was associated with decreased odds (OR 0.71, 95% CI 0.51-0.98; p=0.041) of hospital-acquired pneumonia. Brushing with regular toothbrush and rinsing with chlorhexidine are considered optimal measures of OHC in critically ill patients, but OHC measures are performed inconsistently and differ greatly between individual ICUs. The most effective oral care protocol needs to be elucidated.

Preventive antibiotics may reduce the incidence of infection including pneumonia (van de Beek et al. 2009) but may not improve functional outcome (Westendorp et al. 2012, Westendorp et al. 2015). The effect of beta-blockers, based on the theory of stroke-induced immunodepression, is under investigation (Sykora et al. 2015).

In chronic ICH, some oral medicines, such as angiotensin converting enzyme (ACE) (Sekizawa et al. 1998), amantadine (Nakagawa et al. 1999), and cilostazole (Sinohara 2006), reduce the risk of pneumonia. ACE inhibitors reduce the risk of pneumonia (Caldeira et al. 2012). In patients with previous stroke, risk of pneumonia was lower in those treated with ACE inhibitors compared with control treatment (OR 0.46, 95% CI 0.34-0.62) and angiotensin receptor blockers (ARBs) (OR 0.42, 95% CI 0.22-0.80). This effect was evident especially in an Asian population compared with a non-Asian population.

CONCLUSION

Dysphagia and pneumonia are frequent in patients with acute ICH and are associated with poor outcome. It is estimated that incidence of dysphagia in ICH patients is much higher than in patients with ischemic stroke. Risks and benefits of oral feeding in patients with ICH are unclear due to lack of information. Non-oral feeding strategy may pose a high incidence of pneumonia and mortality. A formal screening procedure for dysphagia and OHC are recommended for reducing the risk of pneumonia in acute ICH. Aggressive interventions to promote oral feeding in the acute stage may improve swallowing.

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Chapter 4

SWALLOWING DYSFUNCTION IN PATIENTS WITH DUCHENNE MUSCULAR DYSTROPHY

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ABSTRACT

Duchenne muscular dystrophy (DMD) is the most common childhood onset muscular dystrophy, with an incidence estimated as 1:3000 live male births. It is an X-linked inherited condition, caused by mutation of the dystrophin gene. Although much of the literature on DMD states that the disease impacts on swallowing function and recommends swallowing assessment as part of their routine examination, the appropriate method of evaluating their swallowing problems had not yet been determined. Recently, we developed an 8-stage Neuromuscular Disease Swallowing Status Scale (NDSSS) and demonstrated its sufficient reliability, validity, and responsiveness in 134 patients with DMD. NDSSS is considered as a useful tool to evaluate swallowing disorders in clinical area, because it is easy to understand and administer by either medical or nonmedical staff. We recommend that their swallowing function should be evaluated periodically and followed up using the NDSSS, and timely consider more detailed assessment with videofluorography.

In this chapter, we reviewed the epidemiology of dysphagia in patients with DMD and the method of evaluating their swallowing problems.

INTRODUCTION

Duchenne muscular dystrophy (DMD) is a recessive X-linked genetic disorder that affects approximately one in 3500 live male births. In DMD, the body is unable to make the protein dystrophin as a result of a large variety of mutations/deletions of the dystrophin gene, found on chromosome Xp21 (Strober, 2000). The condition is characterized by progressive skeletal, respiratory, and cardiac muscle weakness. It leads to severe physical dependency, with loss of ambulation usually between the ages of 10 and 11 years. Death usually occurs in the early 20s, typically from respiratory complications (Finder et al. 2004). However, recent studies have suggested that survival is increasing due to advances in care and, specifically, proactive respiratory management (Hill et al. 2004).

Historically, swallowing issues in DMD have taken a backseat; however, as the life expectancy of patients slowly increases alongside improvements in medical management, such concerns warrant immediate attention. As boys with DMD age, the likelihood that they will become underweight increases. Significant muscle wasting results in weight loss, which is accentuated by eating difficulties experienced at meal times that likely reduce food intake (Davidson and Truby, 2009). It is essential that people with muscle disease maintain adequate nutritional intake, due to the deleterious effects of malnutrition on muscle and ventilatory function (Finder et al. 2004, Hill et al. 2004). Furthermore, any increased risk of aspiration will predispose to respiratory complications in patients with co-existing respiratory muscle weakness, where airway defense mechanisms and cough strength are compromised (Hill et al. 2004, Finder et al. 2004). Jones and colleagues (2016) pointed out that dysphagia can lead to a number of physical, social, and psychological consequences, including poor quality of life, which may be associated with a loss of enjoyment of eating, fear of choking, embarrassment and/or social isolation secondary to coughing, spluttering, and prolonged feeding times. An understanding of the swallowing difficulties experienced by patients with DMD is therefore crucial to enable effective and timely management.

CHARACTERISTICS OF DYSPHAGIA IN PATIENTS WITH DMD

Dysphagia is common in patients with DMD. However, the timing and evolution of this symptom is poorly documented. The prevalence of dysphagia and aspiration in patients with DMD increases with age, and these symptoms can interfere with quality of life (Hull et al. 2012). Dysphagia might be influenced by the severity of impairments in physical function, in addition to age. Wada and colleagues reported significant correlations between scores on the swallowing scales and the severity of physical function scales. According to our experience in our hospital, 15% of DMD patients (age 29.0 ± 7.1 years) who had orally fed began to receive tube feeding within 2 years, and 43% of DMD patients (age 24.0 ± 6.4 years) who reported no symptoms related to swallowing started to show some kind of difficulty eating within 2 years.

Patient self report is useful to medical staff to identify the presence of dysphagia in patients with DMD. The symptoms are as follows: discomfort during swallowing, sensation of food blocked in the throat, difficulty swallowing saliva, coughing during and after meals (van den Engel-Hoek et al. 2013), increased meal duration (Pane et al. 2006), and difficulty starting swallowing (Archer et al. 2013). However, a videofluoroscopic swallow study (VFSS) revealed a higher incidence of swallowing abnormalities than expected from self-report of these symptoms (Hanayama et al. 2008). This suggests that objective evidence is important to increase understanding of the swallowing problems in patients with DMD.

A VFSS reported that swallowing abnormalities were more frequent with solid foods than with jelly or liquids (Hanayama et al. 2008). Common VFSS abnormalities included pooling in the valleculae, pooling in the valleculae after repeated swallowing, pooling in the pyriform sinus, and pooling in the pyriform sinus after repeated swallowing (Hanayama et al. 2008). Nozaki and colleagues (2007) reported that in teenage patients with DMD, swallowing difficulties were more severe during the oral phase than in the pharyngeal phase according to VFSS. Pharyngeal-phase disorders such as pharyngeal residue and decreased pharyngeal contraction were seen more often in patients in their 20s (Nozaki et al. 2007).

It has been proposed that an abnormal swallowing-breathing pattern is related to a stronger respiratory drive in patients with neuromuscular disease (Terzi et al. 2007). However, to the best of our knowledge, there are no reports on the relationship between swallowing function and respiratory function in patients with DMD. We conducted a cross-sectional study to investigate the relationship between swallowing function and respiratory function in patients with DMD.

One hundred and twenty-eight DMD patients (mean age 27.7 ± 7.0 years) who were hospitalized for long-term care at our hospital were evaluated using the Food Intake LEVEL Scale (Kunieda et al. 2013) and were divided into three groups according to their swallowing function: severe dysphagia, moderate dysphagia, and mild dysphagia. Patients were also categorized into three groups based on the type of the respiratory support they received: no ventilation, noninvasive positive pressure ventilation, and tracheostomy positive pressure ventilation.

Of the 128 patients, 109 had the most severe impairments in physical function in activities of daily living, as assessed by Swinyard’s Criteria for Rating Eight Stages of Functional Ability (Swinyard et al. 1957). Table 1 shows the relationship between swallowing function and respiratory function. The three swallowing function groups had almost the same average age. The severe dysphagia group included more tracheostomy positive pressure ventilation patients than the mild dysphagia group did.

This result indicates that swallowing function in patients with DMD may be influenced not only by swallowing muscle weakness due to progressive disease, but also by deterioration of respiratory condition. It should also be considered that a tracheotomy itself might influence swallowing function.

Table 1. The relationship between swallowing function and the use of ventilation in patients with Duchenne muscular dystrophy

	Severe dysphagia	Moderate dysphagia	Mild dysphagia		
Mean (SD) age, years	28.8 (6.9)	27.0 (7.1)	27.4 (7.1)		
No ventilation	0	3	19	$\chi^2 = 31.1$ $p < 0.01$	
NPPV	7	15	23		
TPPV	30	11	20		

NPPV: noninvasive positive pressure ventilation.

TPPV: tracheostomy positive pressure ventilation.

METHODS OF EVALUATING SWALLOWING PROBLEMS IN PATIENTS WITH DMD

Much of the literature on DMD states that the disease impacts swallowing function, and authors recommend swallowing assessment as part of the routine examination (Parker et al. 2005, Finder et al. 2004). Similarly, a Cochrane review in 2004 recommended the establishment of appropriate swallowing assessments for patients with chronic muscle disease (Hill et al. 2004). Proactive management is recommended for patients with DMD and requires appropriate and timely assessment. However, the most appropriate method of evaluating swallowing problems in these patients has not yet been determined (Hill et al. 2004, Jones et al. 2016).

There are some reports of using questionnaires to evaluate swallowing function in patients with DMD. It is important to systematically question patients on their potential symptoms, because patients will often not report them spontaneously (Hanayama et al. 2008). This is an advantage of questionnaires such as the Sydney Swallowing Questionnaire, which assesses the presence of dysphagia based on 17 questions. The Sydney Swallowing Questionnaire is useful because it is simple to conduct and has been validated in patients with DMD (Archer et al. 2013). Hanayama and colleagues (2008) have also evaluated dysphagia in patients with DMD using a simple 11-item questionnaire.

Recently, a universal scale to evaluate dysphagia in various progressive neuromuscular diseases was developed. Common features of patients with progressive neuromuscular diseases include their requirement for foods that are easy to eat because of the difficulty of chewing and their fatigability, and their desire to continue oral intake as long as possible, even in the presence of severe dysphagia, for better quality of life. To address these features, an eight-stage scale, the Neuromuscular disease Swallowing Status Scale (NDSSS), was developed (Wada et al. 2015). The inter- and intra-rater reliabilities, concurrent validity, and responsiveness of the NDSSS have been tested in 134 patients with DMD. Responsiveness was quantified using the standardized response mean. For inter- and intra-rater reliability, the weighted kappa was 0.95 and 1.00, respectively. The NDSSS showed strong correlations with other swallowing assessment tools. The standardized response mean in patients with DMD was higher for the NDSSS than for the other scales. Wada and colleagues (2015) noted that the good responsiveness of the NDSSS for DMD reflects its fine adjustment capabilities. We recommend that swallowing function of patients with DMD be evaluated periodically using the NDSSS, and a more detailed assessment with a VFSS be considered when necessary.

A VFSS and a fiberoptic evaluation of swallowing can be used to confirm the expected diagnosis of dysphagia and quantify the severity of dysphagia (Aloysius et al. 2008). All studies that have used VFSS to investigate dysphagia in patients with DMD have shown that dysphagia was associated with increasingly effortful mastication, reduced hyolaryngeal movement, and poor pharyngeal clearance, all of which can be attributed to progressive oropharyngeal weakness (Shinonaga et al. 2008, Nozaki et al. 2007, Hanayama et al. 2008, Aloysius et al. 2009). When patients with DMD are assessed by VFSS, it is necessary to prepare some food and drinks. Hanayama and colleagues (2008) reported that swallowing abnormalities were observed more frequently with solid foods than with jelly or liquids. In addition, these examinations must take place in a specialized unit where effective cough assistance techniques are available, because of the danger associated with their use in weak DMD patients at risk of aspiration (Toussaint et al. 2016).

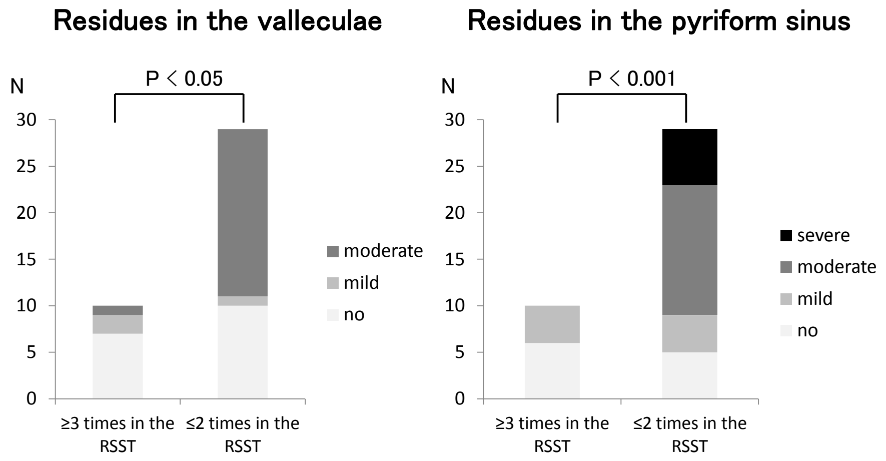

Figure 1

Figure 1. The relationship between score on the repetitive saliva-swallowing test (RSST) and accumulation of residues in the pharynx, as confirmed by a videofluoroscopic swallow study. Patients with Duchenne muscular dystrophy who swallowed three or more times in the RSST showed significantly less accumulation of residues in both the valleculae and the pyriform sinus, as confirmed by a videofluoroscopic swallow study, than patients who swallowed twice or less in the RSST (Mann-Whitney U test).

Surface electromyography might be useful as an auxiliary means of quantifying the severity of dysphagia because it provides a non-invasive, objective method of quantifying muscle activity. Archer and colleagues (2013) reported that dysphagic DMD participants had significantly higher normalized peak electromyography amplitude than healthy control participants for masseter and orbicularis oris muscles.

In addition, the repetitive saliva-swallowing test (RSST) is recommended as a simple bedside swallowing assessment. The RSST has often been used as a screening test for swallowing function (Baba et al. 2008). In this test, patients are asked to sit in a relaxed position and perform repetitive voluntary swallowing as quickly as possible for 30 s. Swallowing three or more times in 30 s is regarded as normal, whereas swallowing twice or less in 30 s is considered impaired. The sensitivity and specificity of the RSST to predict aspiration in videofluorographic examination is 0.98 and 0.66, respectively (Oguchi et al. 2000). However, there are no reports of the RSST being used to evaluate dysphagia in patients with DMD. Therefore, we conducted a cross-sectional study to certify the usefulness of the RSST as an assessment tool in DMD patients with dysphagia.

The RSST was performed by 39 patients with DMD (mean age 20.2 ± 5.3 years, all men). A VFSS was also performed in these participants. Ten of the 39 patients (25.6%) swallowed three or more times in 30 s. Twenty-nine of the 39 patients (74.4%) swallowed twice or less in 30 s, and 23 of these could not swallow their saliva with significant elevation of the larynx. The number of swallows performed in the RSST was not related to aspiration or laryngeal vestibule penetration assessed by the VFSS. However, patients who swallowed three or more times in the RSST showed less accumulation of residues in the pharynx, as confirmed by the VFSS, than patients who swallowed twice or less (Figure 1). Because accumulation of residues is a risk factor for random episodes of silent aspiration, it is important to be able to accurately predict the presence of residues. These results indicate that the RSST can be used to evaluate the severity of dysphagia. When the RSST indicates the presence of residues, a more detailed assessment with VFSS should be considered.

CONCLUSION

We have reviewed the tools available to evaluate dysphagia in patients with DMD. The appropriate management of swallowing problems identified using the tools described in this chapter might prevent aspiration pneumonia and improve nutritional management.

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Cases in head and neck cancer: a multidisciplinary approach

LCCN	2015042630
Type of material	Book
Main title	Cases in head and neck cancer: a multidisciplinary approach / [edited by] Bari Hoffman Ruddy, Henry Ho, Christine Sapienza, Jeffrey J. Lehman.
Published/Produced	San Diego, CA: Plural, [2016]
ISBN	9781597567152 (alk. paper) 1597567159 (alk. paper)
LC classification	RC280.H4
Related names	Hoffman Ruddy, Bari, editor. Ho, Henry, editor. Sapienza, Christine M., editor. Lehman, Jeffrey J., editor.
Contents	Head and neck cancer demographics and team management / Bari Hoffman Ruddy, Jeffrey J. Lehman, Nikhil Rao, Christine Sapienza and Henry Ho -- Lip, anterior floor of mouth and mandibular cancer / Jeff Searl, Terance Ted Tsue, and Douglas A. Girod -- Speech and swallowing after hemiglossectomy with radial forearm free flap reconstruction / Bernice K. Klaben and Keith Casper -- Prolonged dysphagia following chemoradiation of the soft palate and tonsillar fossa / Donna S. Lundy, Mario A. Landera, and Zoukaa B. Sargi -- Overview of multidisciplinary management for oropahryngeal

cancer / Katherine A. Hutcheson, Kristen B. Pytynia, and G. Brandon Gunn -- An interdisciplinary approach to the management of a patient with base of tongue carcinoma / Vrushali Angadi, Thomas J. Gal, and Joseph C. Stemple -- Adenoid cystic carcinoma of the base of tongue / Annette H. May and Elizabeth Feldman -- Minimally invasive approach to treating cancer of the base of tongue / Molly A. Knigge and Timothy M. Mcculloch -- Management of SCCA of the base of tongue / Adam T. Lloyd, Henry Ho, and Erin P. Silverman -- An interdisciplinary approach to the management of HPV positive tonsil cancer with chemo-radiation therapy / Angela L. Campanelli, Jennifer R. Reitz, Wendy D. Leborgne and Matthew R. Garrett -- Overview of multidisciplinary management for hypopharyngeal cancer / Paula A. Sullivan, Ilona Schmalfuss, Charles E. Riggs, Jr., William M. Mendenhall, Elizabeth Leon, and Annette N. Askren; illustrations by Carl C. Askren -- Rare chondrosarcoma of the supraglottis / Jonathon O. Russell, Joseph Scharpf, and Claudio F. Milstein -- Management of SCCA in situ in a professional performer / Adam T. Lloyd, Erin P. Silverman and Brian C. Spector -- Management of recurrent laryngeal cancer / Jeffrey J. Lehman, Vicki Lewis, Erin P. Silverman, and Adam T. Lloyd -- Team approach in treating laryngeal cancer resulting in total laryngectomy / Jennifer Craig, Kyle Mannion, and C. Gaelyn Garrett -- Eliminating aspiration risk through total laryngectomy: a surgical approach requiring laryngectomy / Linda Stachowiak -- Management of nasopharyngeal carcinoma / Jeffrey E. Baylor, Christian E. Soto and Vicki Lewis -- Case of occult primary head and neck SCCA presented with lymphadenopathy / Vicki Lewis, Henry Ho, Aftab H. Patni, Erin P. Silverman, Lee Zehenbot, and Nikhil Rao -- Consideration of communication options in HNC: augmentative and alternative / Laura J. Ball, Jennifer Kent-Walsh, and Nancy A. Harrington --

	Rehabilitation of the laryngectomized individual: alaryngeal communication options / Bari Hoffman Ruddy, Vicki Lewis, and Christine Sapienza -- Patient life experiences essential for humanistic and quality care / Kathleen Ann Kavanagh -- Health literacy implications for head and neck cancer patients / Kristie Hadden, Richard I. Zraick, and Samuel R. Atcherson.
Subjects	Head and Neck Neoplasms--therapy--Case Reports. Head and Neck Neoplasms--diagnosis--Case Reports.
Notes	Includes bibliographical references and index.

Clinical laryngology: the essentials

LCCN	2014007023
Type of material	Book
Main title	Clinical laryngology: the essentials / [edited by] Marvin P. Fried, Melin Tan.
Published/Produced	New York: Thieme, [2014]
ISBN	9781604067491 (hardback: alk. paper)
LC classification	RF510
Related names	Fried, Marvin P., editor. Tan, Melin, editor.
Summary	"Clinical Laryngology addresses the practical issues faced by residents and seasoned practitioners in managing patients with diseases of the larynx. It is a concise manual covering common, everyday procedures that residents must master and provides all the up-to-date information needed by otolaryngologists caring for patients with laryngeal pathology.Key Features: Contains chapters on dysphagia, aspiration, tracheotomy, and cough Includes more than 100 high-quality illustrations and clinical photographs that clarify the information in the text Written and edited by renowned experts in the field of laryngology This clinical resource is the go-to book that residents, fellows, and practicing otolaryngologists will consult when they are in need of practical guidance on managing cases of laryngeal pathology in the course of their practice"--Provided

	by publisher.
Subjects	Laryngeal Diseases. Larynx--pathology.
Notes	Includes bibliographical references.

Communication sciences and disorders: from science to clinical practice

LCCN	2015012337
Type of material	Book
Main title	Communication sciences and disorders: from science to clinical practice / [edited by] Ronald B. Gillam, MD, Thomas P. Marquardt, PhD.
Edition	Third edition.
Published/Produced	Burlington, MA: Jones & Bartlett Learning, [2016]
Description	xvii, 475 pages: illustrations; 24 cm
ISBN	9781284043075 (pbk.: alk. paper) 128404307X (pbk.: alk. paper) 9781284096491
LC classification	RC423 .C647 2016
Related names	Gillam, Ronald B. (Ronald Bradley), 1955- , editor. Marquardt, Thomas P., editor.
Contents	An introduction to the discipline of communication sciences and disorders -- Communication across the life span -- The social and cultural bases of communication -- Speech science -- Developmental speech disorders -- Voice disorders -- Cleft lip and palate -- Fluency disorders -- Motor speech disorders -- Dysphagia -- Language impairment in children -- Acquired neurogenic language disorders -- Hearing science -- Hearing disorders -- Audiologic rehabilitation.
Subjects	Communication Disorders. Communication. Speech--physiology.
Notes	"Navigate 2 Advantage Access" enclosed. Includes bibliographical references and index.

Comprehensive English to Spanish assessment of dysphagia and dysarthria

LCCN	2014941259
Type of material	Book
Personal name	Vitton, Carmen.
Main title	Comprehensive English to Spanish assessment of dysphagia and dysarthria / Carmen Vitton, MS CCC-SLP, Jennifer Maya, MS CCC-SLP, Lindsey Roberts, MS CCC-SLP.
Published/Produced	Dallas, TX: Brown Books Pub. Group, 2014.
ISBN	9781612541976

Diagnosis and evaluation in speech pathology

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Personal name	Haynes, William O.
Main title	Diagnosis and evaluation in speech pathology / William O. Haynes, Rebekah H. Pindzola.
Edition	8th ed.
Published/Created	Upper Saddle River, N.J.: Pearson, c2012.
Description	ix, 422 p.: ill.; 24 cm.
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LC classification	RC423 .H3826 2012
Related names	Pindzola, Rebekah H. (Rebekah Hand)
Contents	Introduction to diagnosis and evaluation: philosophical issues and general guidelines -- Interviewing -- Psychometric considerations in diagnosis and evaluation -- Assessment of children with limited language -- Assessment of school-age and adolescent language disorders -- Assessment of speech sound disorders -- Disorders of fluency -- Assessment of aphasia and adult langauge disorders -- Motor speech disorders, dysphagia, and the oral exam -- Laryngeal and alaryngeal voice disorders -- Assessment of resonance imbalance -- Multicultural issues in assessment -- The diagnostic report.
Subjects	Speech disorders--Diagnosis. Speech disorders--Case studies.

	Speech Disorders--diagnosis.
Notes	Includes bibliographical references (p. 367-409) and index.
Series	The Allyn & Bacon communication sciences and disorders series Allyn & Bacon communication sciences and disorders series.

Diagnosis and evaluation in speech pathology

LCCN	2014048546
Type of material	Book
Personal name	Pindzola, Rebekah H. (Rebekah Hand), author.
Main title	Diagnosis and evaluation in speech pathology / Rebekah H. Pindzola, Auburn University, Laura W. Plexico, Auburn University, William O. Haynes, Auburn University.
Edition	Ninth edition.
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Description	xi, 484 pages: illustrations; 26 cm
ISBN	9780133823905 (Traditional book) 0133823903 (Traditional book)
LC classification	RC423 .H3826 2016
Related names	Plexico, Laura W., author. Haynes, William O., author. Haynes, William O. Diagnosis and evaluation in speech pathology. Preceded by (work):
Contents	Introduction to diagnosis and evaluation: philosophical issues and general guidelines -- Interviewing -- Assessment of children with limited language -- Assessment of school-age and adolescent language disorders -- Assessment of speech sound disorders -- Disorders of fluency -- Assessment of aphasia and adult language disorders -- Motor speech disorders, dysphagia, and the oral exam -- Laryngeal and alaryngeal voice disorders -- Assessment of resonance imbalance -- Multicultural issues in assessment -- The diagnostic report.
Subjects	Speech Disorders--diagnosis.
Notes	Preceded by: Diagnosis and evaluation in speech

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Dysphagia: clinical management in adults and children

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Main title	Dysphagia: clinical management in adults and children / Michael E. Groher, Michael A. Crary.
Edition	Second edition.
Published/Produced	St. Louis, Missouri: Elsevier, [2016]
ISBN	9780323187015
LC classification	RC815.2
Related names	Crary, Michael A., 1952- , author.
Contents	Dysphagia unplugged? -- Normal swallowing in adults? -- Adult neurologic disorders? -- Dysphagia and head/neck cancer? -- Esophageal disorders? -- Respiratory and iatrogenic disorders? -- Clinical evaluation of adults? -- Imaging swallowing examinations: videofluoroscopy and endoscopy? -- Treatment considerations, options, and decisions? -- Treatment for adults? -- Ethical considerations? -- Typical feeding and swallowing development in infants and children? -- Disorders affecting feeding and swallowing in infants and children? -- Evaluating feeding and swallowing in infants and children? -- Treatment of feeding and swallowing difficulties in infants and children?.
Subjects	Deglutition Disorders.

Dysphagia: diagnosis and treatment

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Type of material	Book
Main title	Dysphagia: diagnosis and treatment / Olle Ekberg, editor; foreword by Maximilian F. Reiser.
Published/Created	Heidelberg; New York: Springer, c2012.
Description	xix, 615 p.: ill.; 27 cm.

ISBN	9783642178863 (alk. paper) 3642178863 (alk. paper) 9783642178870 (ebook) 3642178871 (ebook)
LC classification	RC815.2 .D882 2012
Related names	Ekberg, O. (Olle), 1946-
Subjects	Deglutition disorders. Deglutition Disorders--diagnosis. Deglutition Disorders--complications. Deglutition Disorders--therapy.
Notes	Includes bibliographical references and index.
Series	Medical radiology. Diagnostic imaging, 0942-5373 Medical radiology. 0942-5373

Dysphagia: risk factors, diagnosis, and treatment

LCCN	2011042521
Type of material	Book
Main title	Dysphagia: risk factors, diagnosis, and treatment / Brian S. Smith and Mikael Adams, editors.
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Description	xi, 175 p.: ill.; 24 cm.
ISBN	9781619421042 (hardcover)
LC classification	RC815.2 .D96 2012
Related names	Smith, Brian S. (Brian Scott), 1970- Adams, Mikael.
Contents	The management of dysphagia: a clinical and ethical view / David G. Smithard -- Prevention of radiation-induced dysphagia / Piet Dirix and Sandra Nuyts -- Dysphagia in the myopathies / Danielle Ramos Domenis ... [et al.] -- Swallow screening as an essential component of acute stroke management / Julie Luker, Kylie Wall -- Dysphagia: perioperative risk factors, diagnosis, and treatment / P. Bradley Segebarth, Bruce Darden -- The contribution of deglutition sounds for the assessment of dysphagia, aspiration, and penetration: a literature review / Christiane Borr -- Down's syndrome and dysphagia / Tracy Lazenby-Paterson.

Subjects	Deglutition Disorders.
Notes	Includes bibliographical references and index.
Series	Otolaryngology research advances Human anatomy and physiology

Dysphagia assessment and treatment planning: a team approach

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Main title	Dysphagia assessment and treatment planning: a team approach / edited by Rebecca Leonard, Katherine A. Kendall.
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ISBN	9781597565257 (alk. paper) 1597565253 (alk. paper)
LC classification	RC815.2 .D86 2014
Related names	Leonard, Rebecca, editor of compilation. Kendall, Katherine (Staff physician), editor of compilation.
Subjects	Deglutition Disorders--diagnosis. Deglutition Disorders--therapy. Patient Care Planning. Patient Care Team.
Notes	Includes bibliographical references and index.

Functional and GI motility disorders

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Related names	Quigley, Eamonn M. M., editor of compilation. Hongō, Michio, editor of compilation.

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Contents	Oropharyngeal dysphagia and swallowing dysfunction / Sifrim, D.; Vilardell, N.; Clav, P. -- Nonobstructive dysphagia / Hongo, M.; Shoji, T.; Endo, Y. -- Gastroparesis / Parkman, H.P. -- The role of gastric sensorineural dysfunction in functional dyspepsia / Tack, J. -- Chronic intestinal pseudo-obstruction: a neuropathological approach / De Giorgio, R.; Boschetti, E.; Bianco, F.; Cogliandro, R.F.; Volta, U.; Caio, G.; Corinaldesi, R.; Stanghellini, V. -- Small intestinal dysmotility, its role in irritable bowel syndrome and small intestinal bacterial overgrowth: assessment and management / Quigley, E.M.M. -- When and how should we study colonic motility? / Fukudo, S.; Machida, T.; Endo, Y.; Shoji, T.; Kano, M.; Kanazawa, M. -- Difficult defecation: what really helps to assess dysfunction and guide therapy / Corazziari, E.S. -- Functional gastrointestinal disorders: the mind-body dimension / Van Oudenhove, L. -- The role of psychosocial factors in functional gastrointestinal disorders / Lackner, J.M. -- The pharmacology of the gut-brain axis: prospects for future therapeutic progress? / Gabbard, S.; Lacy, B.E. -- Specific clinical contexts. Pt. 1. The intensive care unit / Quigley, E.M.M. -- Specific clinical contexts. Pt. 2. The patient with neurological disease / Choi, M.-G. -- Specific clinical contexts. Pt. 3. The child with neurodevelopmental delay / Carvalho, R.; Di Lorenzo, C. -- Specific clinical contexts. Pt. 4. The postoperative patient / Bassotti, G.; Villanacci, V.; Battaglia, E.; Maurer, C.A.
Subjects	Gastrointestinal Diseases. Gastrointestinal Motility.
Notes	Includes bibliographical references and indexes.
Series	Frontiers of gastrointestinal research, 0302-0665; vol. 33 Frontiers of gastrointestinal research; v. 33. 0302-0665

Handbook of clinical nutrition and stroke

LCCN	2013932865
Type of material	Book
Main title	Handbook of clinical nutrition and stroke / Mandy L. Corrigan, Arlene A. Escuro, Donald F. Kirby, editors.
Published/Produced	New York: Humana Press, [2013]
Description	xxix, 310 pages: illustrations, portraits; 24 cm
ISBN	9781627033794 (alk. paper) 1627033793 (alk. paper)
LC classification	RC388.5 .H362 2013
Related names	Corrigan, Mandy L., editor. Escuro, Arlene A., editor. Kirby, Donald, editor.
Summary	Handbook of Clinical Nutrition and Stroke is a comprehensive reference on nutrition for the multidisciplinary team caring for stroke patients. Targeting physicians, nurse practitioners, clinical dietitians, and advanced allied health and medical students, this volume provides an introduction on the different types of stroke, associated risk factors, and uniquely featured global perspectives on stroke. In addition to discussing stroke risk factors, the book expands upon treatment and management from the acute care setting through rehabilitation, captures the lifespan of patients affected by st. -- Source other than Library of Congress.
Contents	Part I: Introduction to Stroke -- Epidemiology of Stroke -- Types of Strokes -- Stroke Risk Factors -- Perspectives and Approach to Stroke Prevention and Therapy -- Part II: Health-Related Risk Factors for Stroke -- Diabetes Mellitus Prevention and Treatment -- Hypertension/Hyperlipidemia/Hyperhomocysteinemia and Nutrition Approaches -- Obesity and Stroke -- Pediatric/Adolescent Stroke -- Stroke in Younger and Older Adults -- Part III: Medical Management of Stroke -- Medical Management of Stroke -- Malnutrition in Stroke -- Fluid and Electrolyte Management -- Nutrition Support -- Enteral Access --

	Dysphagia in Stroke -- Stroke Nursing Care -- Stroke Rehabilitation -- Ethical Issues in Stroke Patients -- Suggested Stroke Related Resources for the Practitioner and Patient.
Subjects	Cerebrovascular disease--Nutritional aspects--Handbooks, manuals, etc. Cerebrovascular disease--Prevention--Handbooks, manuals, etc. Cerebrovascular disease--Treatment--Handbooks, manuals, etc. Stroke--diet therapy. Nutrition Therapy. Nutritional Physiological Phenomena. Risk Factors. Cerebrovascular disease--Nutritional aspects. Cerebrovascular disease--Prevention. Cerebrovascular disease--Treatment.
Form/Genre	Handbooks, manuals, etc.
Notes	Includes bibliographical references and index.
Series	Nutrition and health Nutrition and health (Totowa, N.J.)

Handbook of gastrointestinal motility and functional disorders

LCCN	2015004809
Type of material	Book
Main title	Handbook of gastrointestinal motility and functional disorders / edited by Satish S.C. Rao, MD, PhD, FRCP, Professor of Medicine, Chief, Gastroenterology/Hepatology, Director, Digestive Health Center, Medical College of Georgia, Georgia Regents University, Henry P. Parkman, MD, Professor of Medicine, Head of GI Motility Laboratory, Gastrointestinal Section, Department of Medicine, Temple University Hospital, Richard W. McCallum, MD, FACP, FRACP (AUST), FAGG, AGAF, Professor of Medicine and Founding Chair, Department of Internal Medicine, Director, Center for Neurogastroenterology and GI Motility, Texas Tech University Health Sciences Center, Paul L. Foster

	School of Medicine.
Published/Produced	Thorofare, NJ: SLACK Incorporated, [2015] ©2015
Description	xviii, 348 pages: illustrations (chiefly color); 26 cm
ISBN	9781617118180 (alk. paper) 1617118184 (alk. paper)
LC classification	RC811 .H36 2015 RC802
Related names	Rao, Satish S. C., editor. Parkman, Henry P., editor. McCallum, Richard W., editor.
Contents	Esophageal symptoms: dysphagia, heartburn, and esophageal chest pain -- Gastroesophageal reflux disease -- Achalasia -- Esophageal spasm, hypercontractile, and hypertensive motility disorders -- Cricopharyngeal disorders -- Esophageal hypersensitivity -- Nausea and vomiting, abdominal pain, postprandial fullness, and early satiety -- Gastroparesis -- Functional dyspepsia -- Dumping syndrome -- Cyclic vomiting syndrome -- Rumination syndrome -- Small intestinal motility symptoms: bloating, distension, and gas -- Chronic intestinal pseudoobstruction -- Small intestinal bacterial overgrowth -- Food intolerance and dietary concepts in functional bowel disorders -- Colonic symptoms: constipation, diarrhea, and fecal incontinence -- Irritable bowel syndrome -- Slow transit constipation -- Dyssynergic defecation and constipation -- Fecal incontinence -- Anorectal disorders: fecal impaction, descending perineum syndrome, rectocele, and levator ani syndrome -- Diabetes and the gut -- Scleroderma and the gut -- Opiate-induced bowel dysfunction.
Subjects	Gastrointestinal system--Motility--Disorders--Handbooks. Gastrointestinal system--Diseases--Handbooks. Gastrointestinal Diseases--Handbooks. Gastrointestinal Motility--Handbooks.
Notes	Includes bibliographical references.

INTRO: a guide to communication sciences and disorders

LCCN	2013033491
Type of material	Book
Personal name	Robb, Michael P., author.
Main title	INTRO: a guide to communication sciences and disorders / Michael P. Robb, PhD.
Edition	Second edition.
Published/Produced	San Diego, CA: Plural Publishing Inc., [2014] ©2014
Description	xiv, 463 pages: illustrations (some color); 27 cm
ISBN	9781597565424 (alk. paper) 1597565423 (alk. paper)
LC classification	RC423 .R57 2014
Variant title	Guide to communication sciences and disorders
Contents	Communications science -- Communication disorders and the professions -- Anatomic processes of speech and hearing -- Child language disorders -- Child phonological disorders -- Fluency disorders -- Cleft lip and palate -- Voice disorders -- Neurogenic communication disorders -- Dysphagia -- Genetics and syndromes -- Hearing disorders -- Auditory rehabilitation.
Subjects	Communicative disorders. Speech therapy. Audiology. Communication Disorders. Hearing Disorders.
Notes	Includes bibliographical references and index.

Principles and practice of palliative care and supportive oncology

LCCN	2012028570
Type of material	Book
Main title	Principles and practice of palliative care and supportive oncology / editors, Ann M. Berger, John L. Shuster, Jr., Jamie Von Roenn.
Edition	4th ed.
Published/Created	Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins, c2013.
Description	xvii, 942 p.: ill.; 28 cm.

ISBN	9781451121278 (hardback: alk. paper) 145112127X (hardback: alk. paper)
LC classification	RC271.P33 P75 2013
Related names	Berger, Ann (Ann M.) Shuster, John L. Von Roenn, Jamie H.
Contents	Difficult pain syndromes: neuropathic pain, bone pain, and visceral pain -- Opioid pharmacotherapy -- Nonpharmacologic management of pain -- Interventional approaches to pain -- Pathology of bone metastases-implications for treatment -- Pathophysiology of chemotherapy-induced peripheral neuropathy -- Cancer-related fatigue -- Fever and sweats -- Hot flashes -- Anorexia/weight loss -- Dysphagia/speech rehabilitation in palliative care -- Chemotherapy-related nausea and vomiting and treatment-related nausea and vomiting -- Assessment of management of chronic nausea and vomiting -- Diarrhea, malabsorption, and constipation -- Bowel obstruction -- Diagnosis and management of effusions -- Hiccups and other gastrointestinal symptoms -- Biology of treatment-induced mucositis -- Oral manifestations and complications of cancer therapy -- Approach to liver metastases in palliative oncology -- Palliative endoscope procedures -- Palliative interventional radiologic procedures -- Pruritus -- Treatment of tumor-related skin disorders -- Dermatologic adverse events during treatment -- Management of pressure ulcers and fungating wounds -- Lymphedema -- Principles of fistula and stoma management -- Dyspnea in the cancer patient -- Hemoptysis, airway obstruction, bronchospasm, cough, and pulmonary complications/symptoms of cancer and its treatment -- Prevention, assessment, and management of treatment-induced cardiac disease in cancer patients -- Management of hypercoagulable states and coagulopathy -- Urologic issues in palliative care -- Impact of hepatic and renal dysfunction on pharmacology of palliative care drugs

	-- Hypercalcemia -- Metabolic disorders in the cancer patient -- Infectious complications/management -- Management of intracranial metastases -- Management of spinal cord and cauda equina compression -- Recognizing and managing delirium -- Treating depression at the end of life -- Anxiety -- Cognitive decline following cancer treatment -- Substance-abuse issues in palliative care -- Epidemiology and prognostication in advanced cancer -- Hospice -- Cultural issues -- Communication during transitions of care -- The family meetings -- Psychosocial consequences of advanced cancer -- Disorders of sexuality and reproduction -- Caregiving in the home -- Management of symptoms in the actively dying patient -- Spirituality -- Bereavement care -- Staff stress and burnout -- Advance directives, decision making, and withholding and withdrawing treatment -- Palliative care: ethics and the law -- Hematologic support of the cancer patient -- Issues in nutrition and hydration -- Complementary, alternative, and integrative therapies in oncology -- Geriatric palliative care -- Palliative care in pediatrics -- Hematopoietic stem cell transplantation and supportive care -- Long-term survivorship: late effects -- Survivorship issues in adults -- Outcomes assessment in palliative care -- Measurement of quality of life outcomes -- Research issues: ethics and study design.
Subjects	Cancer--Palliative treatment. Cancer--Complications--Treatment. Cancer pain--Treatment. Neoplasms--therapy. Neoplasms--complications. Pain Management--methods. Palliative Care--methods.
Notes	Includes bibliographical references and index.

Principles of deglutition: a multidisciplinary text for swallowing and its disorders

LCCN	2012943384
Type of material	Book
Main title	Principles of deglutition: a multidisciplinary text for swallowing and its disorders / Reza Shaker ... [et al.], editors.
Published/Created	New York: Springer, c2013.
Description	xix, 1017 p.: ill. (some col.); 27 cm.
ISBN	9781461437932 (alk. paper) 1461437938 (alk. paper)
LC classification	RC815.2 .P75 2013
Related names	Shaker, Reza.
Partial contents	General Aspects of Deglutition. Overview of Deglutition and Digestion; Gustation, Olfaction and Deglutition; Coordination of Deglutition and Respiration; Airway Protective Mechanisms, Reciprocal Physiology of the Deglutitive Axis -- Central Control of Deglutition. Cerebral Cortical Control of Deglutition; Brainstem Control of Deglutition: Swallowing Pattern Generator; Brainstem Control of Deglutition: Brainstem Neural Circuits and Mediators Regulating Swallowing -- Preparatory/Oral Phase of Deglutition. Oral Phase Preparation and Propulsion: Anatomy, Physiology, Rheology, Mastication and Transport; Deglutitive Oral Pressure Phenomena; Effect of Aging on the Oral Phase of Deglutition; Nascent Oral Phase -- Pharyngeal Phase of Deglutition. Development, Anatomy and Physiology of the Pharynx; Development, Anatomy and Physiology of the Larynx; Development, Anatomy and Physiology of the Lungs; Effect of Aging of the Pharynx and the UES; Nascent Pharynx, Physiology, Reflexes -- UES and its Deglutitive Function. Development, Anatomy and Physiology of the Upper Esophageal Sphincter and Pharyngo-Esophageal Junction; Deglutitive Pharyngeal and UES Pressure Phenomena -- Esophageal Phase of Deglutition. Development,

Anatomy and Physiology of the Esophagus; Normal Aging and the Esophagus; Nascent Esophagus, Sensory-Motor Physiology during Maturation -- Esophageal Motility and Its Deglutitive Function. Esophageal Motor Physiology; Sphincter Mechanisms at the Esophago-Gastric Junction; Motility and Pressure Phenomena of the Esophagus -- Oral/Pharyngeal Phase Dysphagia. Symptom Indices for Dysphagia Assessment and Management; Cerebro-Vascular Accidents and Dysphagia; Progressive Neurologic Disease and Dysphagia (including Parkinson's Disease, Multiple Sclerosis, Amyotrophic Lateral Sclerosis, Myasthenia Gravis, Post Polio Syndrome); Disorders of Appetite, Eating and Swallowing in the Dementias; Dystrophies and Myopathies (including Muscular, Oculopharyngeal); Dysphagia Secondary to the Effects of Chemo and Radiation Therapy; Swallowing Syncope; Globus Pharyngeus; Deglutition in Patients with Tracheostomy, Nasogastric Tubes and Orogastric Tubes; Dysphagia Secondary to Systemic Diseases -- Upper Esophageal Sphincter Opening Dysfunction. Zenker's Diverticulum; Cricopharyngeal Bar; Cricopharyngeal Achalasia; UES Opening Muscle Dysfunction -- Esophageal Phase Dysphagia: Motor Disorders. Achalasia and Ineffective Esophageal Motility; Esophageal Spasm/Non-Cardiac Chest Pain Hypertensive Esophageal Peristalsis (Nutcracker Esophagus) Hypertensive Lower Esophageal Sphincter -- Esophageal Phase Dysphagia: Structural Abnormalities. Benign and Malignant Tumors of the Esophagus; Strictures, Rings, Webs (peptic, caustic, radiation, anastomotic); Esophageal Foreign Bodies: Food Impaction and Foreign Bodies; Post-Surgical Dysphagia: Post-Nissen Fundoplication, C-spine surgery, Thyroid surgery, Gastric Banding, Gastric Bypass; Drug-induced Esophageal Injury; Infectious Esophagitis; Eosinophilic Esophagitis; Gastroesophageal Reflux Disease; Barrett's

	Esophagus; Extraesophageal Manifestations of Reflux Disease and Dysphagia; Hiatal Hernia -- Eating Disorders. Psychogenic Dysphagia; Rumination Syndrome -- Treatment Options for Deglutition Disorders. Compensatory Management and Treatment in Dysphagia; Rehabilitative Treatments; Electrical Stimulation Treatment; Pharmacotherapy of UES Spastic Disorder; Open and Endoscopic Cricopharyngeal Myotomy; Surgical Treatment of Zenker's Diverticulum; Dilatations (UES, Esophagus, LES) Balloon Dilations, Bougies; Esophageal Stenting for Relief of Dysphagia; Botulinum Toxin for LES Spastic Disorders; Surgical Treatment of Achalasia and Spastic Esophageal Disorders; Vocal Fold Medialization, Arytenoid Adduction, and Partial Pharyngectomy; Laryngohyoid Suspension; Surgical Management of Life-Threatening Aspiration; Anti-Reflux Surgery; Tube Feeding: Indications, Considerations and Technique.
Subjects	Deglutition disorders. Deglutition. Deglutition Disorders--therapy. Deglutition--physiology. Deglutition Disorders--physiopathology.
Notes	Includes bibliographical references and index.

Stroke syndromes

LCCN	2011042775
Type of material	Book
Main title	Stroke syndromes / edited by Louis R. Caplan, Jan van Gijn.
Edition	3rd ed.
Published/Created	Cambridge; New York: Cambridge University Press, 2012.
Description	x, 621 p.: ill. (some col.); 29 cm.
ISBN	9781107018860 (hardback)
LC classification	RC388.5 .S8567 2012
Related names	Caplan, Louis R. Van Gijn, J.

Summary	"The first two editions of Stroke Syndromes were widely welcomed as authoritative reference works in the assessment and diagnosis of stroke. This revised and updated third edition remains the definitive guide to patterns and syndromes in stroke. A comprehensive survey of all types of neurological, neurophysiological and other clinical dysfunction due to stroke. The book contains descriptions of clinical problems encountered in stroke patients and their differential diagnosis, enhancing pattern recognition and enabling clinicians to differentiate between possible locations on the basis of symptoms and signs. The companion volume Uncommon Causes of Stroke completes this highly authoritative reference work which clinicians in neurology will find essential to the understanding and diagnosis of stroke"--Provided by publisher.
Contents	Machine generated contents note: Introduction; Part I. Clinical Manifestations: 1. Hemiparesis and other types of motor weakness Adria Arboix and Josep Marti-Vilalta; 2. Sensory abnormalities Jung S. Kim; 3. Cerebellar ataxia Chris Diener and Dagmar Timmann; 4. Cognitive and behavioral abnormalities secondary to cerebellar stroke Jeremy Schmahmann; 5. Headache: stroke symptoms and signs Conrad Estol; 6. Eye movement abnormalities Shirley Wray and Louis R. Caplan; 7. Cerebral visual dysfunction Jarson Barton and Louis R. Caplan; 8. Visual symptoms Shirley Wray; 9. Vestibular syndromes and vertigo Marianne Dieterich and Thomas Brandt; 10. Auditory disorders in stroke Robert Baloh; 11. Abnormal movements Joseph Ghika; 12. Seizures and stroke Chris Bladin and Geoff Donnan; 13. Disturbances of sleep Claudio Bassetti; 14. Coma and decreased levels of consciousness Eelco Wijdicks; 15. Aphasia and stroke Argye Hillis and Elisabeth Marsh; 16. Agitation and delirium John Brust and Louis R. Caplan; 17. Frontal lobe stroke syndromes Randolph Marshall; 18. Memory loss Jose Ferro and Isabel

Martins; 19. Neurobehavioral aspects of deep hemisphere stroke Jose Ferro and A. Verdelho; 20. Right hemisphere syndromes Elliott Ross and Louis R. Caplan; 21. Post-stroke dementia Dider Leys and Florence Pasquier; 22. Disorders of mood and behavior Randolph Marshall; 23. Agnosias, apraxias and callosal disconnection syndromes Arnaud Saj and Patrik Vuilleumier; 24. Muscle and peripheral nerve manifestations of stroke Rick Logigian and Pariwat Thaisettawatkul; 25. Cardiac and autonomic manifestations of stroke E. Camargo and M. Samuels; 26. Dysarthria Edward Feldmann; 27. Dysphagia and aspiration syndromes Sandeep Kumar; 28. Respiratory dysfunction F. Vingerhoets; Part II. Vascular Topographic Syndromes: 29. Arterial territories of the human brain Laurent Tatu, Thierry Moulin, Fabrice Vuillier and Julien Bogousslavsky; 30. Middle cerebral arteries J. P. Mohr; 31. Anterior cerebral artery John Brust; 32. Anterior choroidal artery territory infarcts Cathy Helgason and Louis R. Caplan; 33. Thalamic infarcts and hemorrhages Michel Barth and Louise R. Caplan; 34. Caudate infarcts and hemorrhages Chin-Sang Chung and Louis R. Caplan; 35. Posterior cerebral artery C. Chaves and Louis R. Caplan; 36. Large and panhemispheric infarcts R. Kollmar, J. Bosel and S. Schwab; 37. Multiple and bihemispheric infarcts Emre Kumral; 38. Midbrain infarcts M. Hommel and Louis R. Caplan; 39. Pontine infarcts and hemorrhages Chin-Sang Chung and Louis R. Caplan; 40. Medullary infarcts and hemorrhages Jung S. Kim; 41. Cerebellar infarcts Mikael Mazighi and P. Amarenco; 42. Border zone infarcts E. Bernd Ringelstein, Ralph Dittrich and Dittrich Stogbauer; 43. Lacunar syndromes J. Bamford; 44. Basal ganglia hemorrhages Chin-Sang Chung, Kwang-Yeol Park and Louis R. Caplan; 45. Lobar hemorrhages Carlos Kase; 46. Intraventricular hemorrhages D. Hanley and W. Ziai; 47. Subarachnoid hemorrhage syndromes Jan van Gijn

	and G. Rinkel; 48. Brain venous thrombosis syndromes Jerome Mawet, Isabelle Crassard and Marie-Germaine Bousser; 49. Carotid occlusion syndromes Seemant Chaturvedi; 50. Cervical artery dissection syndromes Marcel Arnold, Heinrich P. Mattle and Mathias Sturzenegger; 51. Intracranial artery dissections C. Estol and Louis R. Caplan; 52. Syndromes related to large artery thromboembolism in the posterior circulation Louis R. Caplan; 53. Spinal stroke syndromes Mathias Sturzenegger; Index.
Subjects	Cerebrovascular disease--Diagnosis. Diagnosis, Differential. Symptoms. MEDICAL / Neurology.

Surgery on the move

LCCN	2012276436
Type of material	Book
Personal name	Morgan, Jenna, author.
Main title	Surgery on the move / by Jenna Morgan, Harriet Walker, Andrew Viggars.
Published/Produced	Boca Raton, Florida CRC Press, 2013 ©2014
Description	xv, 347 pages: colour illustrations; 22 cm.
ISBN	9781444176018 (pbk) 1444176013 (pbk) 9781482208610 (ePub ebook) 148220861X (ePub ebook) 1482208628 (ebook) 9781482208627 (ebook)
Related names	Walker, Harriet, author. Viggars, Andrew, author.
Contents	Machine generated contents note: pt. I General surgery -- 1.Pre-operative care -- 1.1.Pre-operative assessment in the elective patient -- 1.2.Pre-operative assessment in the emergency surgical patient -- 1.3.How to manage conditions of special relevance -- 1.4.Preparation for theatre -- 1.5.First time in the

operating theatre -- 1.6.Incisions -- 2.Lumps and bumps -- 2.1.Abdominal wall herniae -- 2.2.How to examine for groin herniae -- 2.3.Benign soft tissue swellings -- 2.4.Malignant soft tissue swellings -- 2.5.How to examine a soft tissue swelling -- 2.6.Superficial infections -- 2.7.Skin cancers -- 3.Breast -- 3.1.Breast examination -- 3.2.Benign breast diseases -- 3.3.Breast cancer -- 3.4.Breast screening -- 4.Endocrine -- 4.1.Examination of the thyroid gland -- 4.2.Hyperthyroidism -- 4.3.Hypothyroidism -- 4.4.Carcinoma of the thyroid gland -- 4.5.Primary hyperparathyroidism -- 4.6.Phaeochromocytoma -- 4.7.Conn's syndrome (primary hyperaldosteronism) -- 4.8.Cushing's syndrome -- 4.9.Multiple endocrine neoplasia (MEN) -- 5.Hepatobiliary -- 5.1.Hepatobiliary anatomy -- 5.2.Upper abdominal pain -- 5.3.How to examine the abdomen -- 5.4.Jaundice: pathophysiology and classification -- 5.5.Gallstones -- 5.6.Acute pancreatitis -- 5.7.Pancreatic malignancies -- 5.8.Primary hepatic malignancies -- 5.9.Secondary hepatic malignancies -- 5.10.Biliary malignancies: gallbladder -- 5.11.Biliary malignancies: cholangiocarcinoma -- 6.Upper GI -- 6.1.Gastro-oesophageal reflux disease (GORD) -- 6.2.Hiatus hernia -- 6.3.Achalasia -- 6.4.Mallory--Weiss tear -- 6.5.Oesophageal varices -- 6.6.Oesophageal cancer -- 6.7.How to manage acute upper GI haemorrhage -- 6.8.How to manage acute dysphagia -- 6.9.Peptic ulcer disease -- 6.10.Stomach cancer -- 6.11.Intestinal ischaemia -- 6.12.Small bowel obstruction -- 6.13.Small bowel tumours -- 7.Colorectal -- 7.1.Anatomy of the bowel -- 7.2.Appendicitis -- 7.3.How to manage a case of suspected appendicitis -- 7.4.Inflammatory bowel disease -- 7.5.Crohn's disease -- 7.6.Ulcerative colitis -- 7.7.Ischaemic colitis -- 7.8.Infective colitis -- 7.9.Irritable bowel syndrome -- 7.10.Colorectal cancer -- 7.11.Colonic polyps -- 7.12.Diverticular disease -- 7.13.Angiodysplasia --

7.14.Endoscopy -- 7.15.Pilonidal disease -- 7.16.Haemorrhoids (commonly referred to as `piles') -- 7.17.Rectal prolapse -- 7.18.Anal fissure -- 7.19.Anorectal abscess -- 7.20.Fistula-in-ano -- 7.21.Anal cancer -- 7.22.Stomas -- 8.Vascular -- 8.1.Vascular anatomy -- 8.2.AAA -- 8.3.How to manage a suspected leaking AAA -- 8.4.Aortic dissection -- 8.5.Acute limb ischaemia -- 8.6.How to manage a case of possible ALI -- 8.7.Chronic lower limb ischaemia -- 8.8.How to examine the peripheral vascular system -- 8.9.The diabetic foot -- 8.10.Varicose veins -- 8.11.How to examine varicose veins -- 8.12.Carotid disease -- 9.Acute urology -- 9.1.How to examine the male genitalia -- 9.2.Testicular torsion -- 9.3.Epididymo-orchitis -- 9.4.Lower urinary tract infection (pyelonephritis) -- 9.5.Upper UTI (pyelonephritis) -- 9.6.Fournier's gangrene -- 9.7.Urolithiasis -- 9.8.Urinary retention -- 9.9.Frank haematuria -- 9.10.Paraphymosis -- 9.11.Priapism -- 9.12.How to insert a urinary catheter -- 10.Burns and trauma -- 10.1.ATLS principles -- 10.2.Abdominal trauma -- 10.3.Thoracic trauma -- 10.4.Head trauma -- 10.5.Burns -- 11.Post-operative care -- 11.1.Post-operative care on the surgical ward -- 11.2.General post-operative complications -- pt. II Self-assessment -- 12.Pre-operative care -- 12.1.Pre-operative care EMQs -- 12.2.Pre-operative care EMQ answers -- 12.3.Pre-operative care SBAs -- 12.4.Pre-operative care SBA answers -- 13.Lumps and bumps -- 13.1.Lumps and bumps EMQs -- 13.2.Lumps and bumps EMQ answers -- 13.3.Lumps and bumps SBAs -- 13.4.Lumps and bumps SBA answers -- 14.Breast -- 14.1.Breast EMQs -- 14.2.Breast EMQ answers -- 14.3.Breast SBAs -- 14.4.Breast SBA answers -- 15.Endocrine -- 15.1.Endocrine EMQs -- 15.2.Endocrine EMQ answers -- 15.3.Endocrine SBAs -- 15.4.Endocrine SBA answers -- 16.Hepatobiliary -- 16.1.Hepatobiliary EMQs -- 16.2.Hepatobiliary EMQ answers --

	16.3.Hepatobiliary SBAs -- 16.4.Hepatobiliary SBA answers -- 17.Upper GI -- 17.1.Upper GI EMQs -- 17.2.Upper GI EMQ answers -- 17.3.Upper GI SBAs -- 17.4.Upper GI SBA answers -- 18.Colorectal -- 18.1.Colorectal EMQs -- 18.2.Colorectal EMQ answers -- 18.3.Colorectal SBAs -- 18.4.Colorectal SBA answers -- 19.Vascular -- 19.1.Vascular EMQs -- 19.2.Vascular EMQ answers -- 19.3.Vascular SBAs -- 19.4.Vascular SBA answers -- 20.Urology -- 20.1.Urology EMQs -- 20.2.Urology EMQ answers -- 20.3.Urology SBAs -- 20.4.Urology SBA answers -- 21.Trauma and burns -- 21.1.Trauma and burns EMQs -- 21.2.Trauma and burns EMQ answers -- 21.3.Trauma and burns SBAs -- 21.4.Trauma and burns SBA answers -- 22.Post-operative care -- 22.1.Post-operative care EMQs -- 22.2.Post-operative care EMQ answers -- 22.3.Post-operative care SBAs -- 22.4.Post-operative care SBA answers.
Subjects	Surgery.
Notes	Includes index.
Series	Medicine on the move

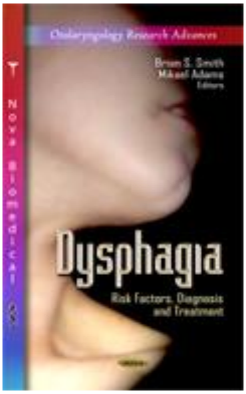
Swallow safely: how swallowing problems threaten the elderly and others. a caregiver's guide to dysphagia: recognition, treatment, and prevention

LCCN	2016934860
Type of material	Book
Personal name	Sayadi, Roya.
Main title	Swallow safely: how swallowing problems threaten the elderly and others. a caregiver's guide to dysphagia: recognition, treatment, and prevention / Roya Sayadi, Joel Herskowitz.
Edition	2nd edition.
Published/Produced	Natick, MA: Inside/Outside Press, 2016.
ISBN	9780981960159 (pbk.)

RELATED NOVA PUBLICATIONS

DYSPHAGIA: RISK FACTORS, DIAGNOSIS AND TREATMENT

Editors: Brian S. Smith and Mikael Adams


Otolaryngology Research Advances
Brian S. Smith
Mikael Adams
Editors
Nova Biomedical
Dysphagia
Risk Factors, Diagnosis and Treatment

ISBN: 978-1-61942-104-2 **Publication Date:** 2012

Swallowing difficulties or dysphagia, can occur with numerous conditions, some of which are permanent, some reversible, and some progressive. The underlying etiology can be neurological, muscular, or mechanical/obstructive. The authors of this book present topical research in the study of the risk factors, diagnosis and treatment of dysphagia. Topics discussed include the etiology and prognosis associated with dysphagia; prevention of radiation-induced dysphagia; dysphagia in the myopathies; swallow screening as an essential component of acute stroke management and the perioperative risk factors for dysphagia. (Imprint: Nova Biomedical)

DYSPHAGIA ON THE ELDERLY: RISK FACTORS, DIAGNOSIS AND TREATMENT[*]

P. Santoro and F. Cardoso
University Castelo Branco, Strictusensu Program in Human Movement
Science of Science in Human Movement, LABFILC,
Laboratory Philosophical Issues in Applied Knowledge, Recreio dos
Bandeirantes, Rio de Janeiro, Brazil

This chapter provides a reflection of some representative aspects of the problem of dysphagia in the elderly, this will be operationalized by means of literature review, understanding this strategy as being very appropriate in order to review, analyze, interpret and criticize theoretical considerations, even create new paradigms and propositions of explanation and understanding of phenomena from all branches of knowledge that can support a further deepening of the studies by experimental investigations or even descriptive. The location and selection of content data of the study were performed in master's theses, books and scientific articles on the subject, and from research sites in open access to international journals and virtual libraries of institutions of higher education. Therefore this chapter is divided into stages emphasizing the concept, impact, primary and secondary traits peculiar to senescence, the criteria for differential diagnosis and possibilities for strategies and interventions.

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[*] The full version of this chapter can be found in *Advances in Medicine and Biology. Volume 49*, edited by Leon V. Berhardt, published by Nova Science Publishers, Inc, New York, 2012.

RELATIONSHIP BETWEEN DYSPHAGIA AND DEPRESSIVE STATES IN PATIENTS WITH PARKINSON'S DISEASE*

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Aspiration pneumonia related to dysphagia is known to be the leading cause of death in patients with Parkinson’s disease (PD). We investigated the relationship between depressive states and dysphagia in patients with PD. A hundred and twenty-seven PD patients gave their informed consent and were enrolled in this study. We used the Beck Depression Inventory II (BDI-II) questionnaire to determine the participants’ depressive states, and also used a questionnaire to assess participants’ state of dysphagia. Participants were divided into four groups according to their BDI-II score. We compared the PD patients with Swallowing Disturbances Questionnaire (SDQ) scores of more than or equal to 11 with the SDQ scores of less than 11 regarding depressive categories. A logistic regression analysis was conducted to calculate odds ratios (ORs) and their 95% confidence intervals (95%CI) adjusting for age, sex, disease duration, wearing-off phenomenon and severity of movement disorder. OR (95%CI) of depressive categories, in which the trivial class was

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[*] The full version of this chapter can be found in *Parkinson's Disease: Diagnosis, Treatment and Prognosis*, edited by Chiyo Yoshida and Ami Ito, published by Nova Science Publishers, Inc, New York, 2012.

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set as a reference group, were 3.28 (0.93-11.55), 13.44 (3.10-58.16), 30.35 (5.65-162.97) in the mild class, the moderate class and the severe class, respectively. This study suggests that there may be a strong relationship between depressive states and dysphagia in patients with PD.

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